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SUDDEN CARDIAC DEATH *

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Unexpected sudden death due to heart disease occurs frequently. It is the purpose of this discussion to review the pathological physiology, the clinical causes and aspects of sudden cardiac death, and to stimulate further interest in those cases where immediate therapy can be life saving. The definition of sudden death as used here will imply death that occurs usually within a few minutes but no longer than twelve hours after incapacitating symptoms.

PATHOLOGICAL PHYSIOLOGY

The cause of cardiac death has been divided by Hellerstein [1] into two categories; namely, mechanism death and muscle death. Mechanism death is a disturbance in the coordinated beat of the heart, such as ventricular fibrillation or cardiac standstill. When acute myocardial infarction is found at autopsy, it is obvious that a disturbance in the coordinated beat has occurred. Had the coordinated beat not been destroyed, the heart would have continued beating. Muscle death, on the other hand, refers to those cases in which extensive degenerative changes develop in the myocardium leading to cardiomegaly and heart failure.

Abnormalities of rhythm following an acute coronary artery occlusion have been attributed to an irritable focus in the infarcted area from which arise ectopic stimuli resulting in disturbances in cardiac rhythm and frequently in fatal ventricular fibrillation. According to Brofman [2] et al., a difference in oxygenation of two adjacent areas of myocardium, associated with a difference in electrical potential across the transitional zone, acts as a "trigger" to destroy the coordinated mechanism of the heart, thereby inducing ventricular fibrillation. Milch [3] and his group working with dogs concluded that the animals were protected from ventricular fibrillation following coronary occlusion by thoracic sympathectomy and procaine injection of the stellate ganglia. The fatal stimulus from an extra corporeal source was thought to have been mediated predominantly over the cardiac sympathetic plexus which arises from the cervical and thoracic sympathetic ganglia. When the heart was ischemic and irritable, it seemed receptive to almost any type of somatic stimulus. Such a stimulus might initiate an abnormality of cardiac rhythm and frequently result in fatal ventricular arrhythmias.

CLINICAL CAUSES OF SUDDEN CARDIAC DEATH

Among natural causes of sudden death, heart disease is by far the most common. In a series described by Bedford,[4] cardiovascular causes accounted for 73 per cent of all deaths; in Sheldon's [5] 443 cases of sudden death, heart disease was responsible for 243 deaths. Hammann,[6] after reviewing several other authors' series of cases, concluded that diseases of the cardiovascular system, including arterial embolism, thrombosis, and hemorrhage as a cause of sudden

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death, accounted for 91 per cent of cases of which 65 per cent were due to disease of the coronary arteries, 21 per cent having occurred with valvular heart disease, 10 per cent with myocardial disease, and 3 per cent with cardiac hypertrophy. Rabson and Helpern [7] found that diseases of the heart and aorta were responsible for 45 per cent of cases of sudden death. There being sometimes an overlap, several causes for sudden unexpected death are present, e.g., a patient with angina may also have aortic stenosis and die from heart block. As is well known, many cardiac patients die suddenly and unexpectedly from pulmonary embolism which is the result of an embolus arising from one of the deep calf or plantar veins and the cause of death is not heart disease per se.

CORONARY ARTERY DISEASE

Sudden death is due most commonly to coronary artery disease which accounts for at least 60 to 80 per cent of all unexpected cardiac deaths.[8] Boemke [9] analysed 1,816 cases of sudden death in soldiers during World War II of which 1,217 sudden cardiac deaths were due to coronary sclerosis. Similarly, Monitz and Zamcheck [10] reviewed 1,000 cases of sudden deaths in apparently healthy young soldiers between the ages of 18 and 40 during the period 1942 to 1946. Of 350 cases of heart disease in this series, 300 deaths were due to coronary artery disease. Thus, if an adult apparently in perfect health suddenly falls dead, the chances are overwhelmingly in favor of coronary artery disease. In such a series of cases reported by Cairns,[11] a point of great interest was the number in which the main finding was diseased coronary arteries without thrombosis or occlusion. The arteries revealed coronary atherosclerosis with pronounced coronary narrowing, but no occlusion or infarction. French and Dock [12] observed coronary narrowing without infarction in many cases of sudden death in young soldiers. Levy and Bruenn [13] described a series of hospitalized patients with coronary heart disease who died suddenly, revealing at autopsy coronary atherosclerosis, but no fresh occlusion or other anatomic lesion to explain the immediate cause of death. They observed an 11.8 per cent incidence of sudden death in a group of patients with coronary sclerosis without thrombosis as compared with 33.3 per cent in a similar group with coronary thrombosis. Helpern and Rabson,[7] too, carefully analysed 2,030 consecutive cases of sudden and unexpected death verified at autopsy, coronary atherosclerosis accounting for 30.4 per cent of the entire series. Of 617 instances of coronary artery sclerosis, almost three-quarters (73.3 per cent) had no grossly demonstrable associated fresh thrombosis of the vessels. In most cases atheroma, with or without calcification, had produced significant diminution of luminal calibre, myocardial infarction having occurred in only 5 per cent.

These various series of reports seem to indicate that death from coronary thrombosis is less common than is clinically believed. As was expected, the most fatal years were between the ages of forty and fifty-four, most of the deaths occurring in white men. Negroes were certainly not immune to sudden death from coronary disease; however, of 46 cases of death attributable to obstruction of coronary ostia in syphilitic aortitis, 39 per cent were negroes.

Coronary embolism is relatively uncommon but obviously can produce sudden unexpected death. Nearly all reported instances have been due to detachment of part of a vegetation resulting from bacterial endocarditis.

CARDIAC RUPTURE

Rupture of the heart is one of the common causes of sudden death in the first few weeks after acute myocardial infarction. Martland [8] found that rupture of the heart comprised about 7 per cent of the 318 cases of coronary occlusion in his series, while Helpern and Rabson [7] reported an incidence of 10 per cent. Of clinical interest in Cairns'[11] series was the fact that of the 17 cases of coronary thrombosis with myocardial infarction, 14 died of a rupture of the infarct. In all of these cases, not only was the cardiac infarct unsuspected, but the deceased had not been considered to be ill at all prior to death. Jetter and White [14] found that, out of 22 cases in men-

tal institutions dying with a myocardial infarct, 16 had died with cardiac tamponade. None of these cases had given a complaint or symptom suggesting cardiac infarction, a fact which would suggest that the period of bed rest recommended for the diagnosed case of coronary thrombosis has much to commend it.

Rupture of the heart occurs most frequently in the first week, sometimes in the second week, and usually results from a tear through the infarcted area which is still necrotic and soft. Softening of the heart muscle is a major factor, yet Edmondson and Hoxie [15] found that patients who had persistent hypertension after infarction were three times more likely to develop cardiac rupture than those who had normal or hypotensive blood pressures. Cardiac rupture depends to a great extent also on the site and extent of the infarct and is favored by absence of fibrosis in the infarct.

Although cardiac infarction due to coronary artery disease is the most common cause of rupture of the heart, the latter may result rarely from bacterial endocarditis, myocardial abscess due to sepsis, dissecting aneurysm of the sinus of Valsalva, gummatous myocarditis, tuberculous perimyocarditis, echinococcus cyst and malignancy.

DISEASES OF THE AORTA

In cardiovascular syphilis, syphilitic aortitis is almost exclusively the lesion found. This condition can lead to sudden death, according to Oram,[16] in one of four ways; namely, by the syphilitic process occluding the coronary artery ostia, by rupture of a saccular aneurysm, by the development of aortic incompetence which interferes with filling of the coronary arteries, and by rupture of a focal dissecting aneurysm which usually ruptures into the pericardium.

Pick [17] described a case of dissecting aneurysm of the aorta which extended to the aortic root and resulted in hemorrhage into the atrial septum involving the area of the A-V node. The dissection eventually led to sudden death with rupture into the pericardium. According to Shennan,[18] approximately 10 per cent of all cases of dissecting aneurysm recover from the initial attack, as a result of the dissection rupturing back into the lumen of the aorta.

Oram and Holt [19] reviewed the various ways in which the coronary arteries can be involved in dissecting aneurysm, the more important of which include occlusion of the coronary ostia by the aneurysm, extension of the dissection along the coronary arteries, and compression of the coronary arteries from without by extravasated blood from the aneurysm.

Sudden death from rupture of the aorta which may be hypoplastic in coarctation of the aorta occurred in 19 per cent of Abbott's [20] cases. Sudden death may occur at the operating table from asystole when the aorta is clamped, or later from the tearing out of sutures from a hypoplastic aorta distal to the constriction, or from rupture along the suture line, owing to the nonhealing of the anastomosis because of superimposed infection.

Among 5000 necropsies performed by the Vienna Medico Legal Institute, according to Jolk,[21] only 4 instances of advanced stenosis of the aortic isthmus were encountered. Recently he reported such a case of sudden and unexpected death in a young athlete who had just finished weightlifting and wrestling. The stenosis, having caused a mechanical obstruction with considerable dilatation of the aorta above the isthmus, meant immediate death as a result of the rupture of the aorta. It was felt that the physiological rise in blood pressure accompanying such physical exercise very definitely exerted a deleterious influence.

DISEASES OF THE MYOCARDIUM

As is generally known, myocarditis may occur as a complication of any acute infectious process brought on by a wide variety of etiologic agents, such as bacteria, fungi, viruses, yeasts, Rickettsia, spirochetes, protozoa, and round worm infections. The clinical diagnosis of myocarditis is usually not difficult if the physician is aware of its existence and suspects its presence. Not infrequently, frank cardiac failure with edema will occur with its attendant clinical picture, various arrhythmias being en-

countered. An unexplained bradycardia or tachycardia out of all proportion to the associated disease suggests myocardial involvment. Dyspnea, cyanosis, and low blood pressure may be prominent; the electrocardiogram may be abnormal. The course then is generally rapid, and sudden death is not unusual. On the other hand, the illness may last for months; and recovery may take place after there has been serious involvment of the heart muscle.

Unexpected death occurs quite often in myocarditis. Gore and Saphir [22] reported 35 fatal cases of myocarditis of which there were 15 sudden and unexpected cardiac deaths. Boemke,[9] having analysed 1,816 cases of sudden death in soldiers during World War II, found 94 cases of sudden death due to myocarditis. Karni [23] studied 8 cases of isolated myocarditis, acute and chronic, which developed cardiac failure and died suddenly and unexpectedly. Bowden,[24] in a careful search for the cause of death in babies found dead in bed, noted five examples of acute myocarditis in a consecutive series of 50 babies. Among 60 children with myocarditis whom Saphir [25] studied, 9 died unexpectedly; 5 other children died suddenly with myocarditis while ill with laryngotracheobronchitis; and 3 patients with encephalomyocarditis died unexpectedly. Saphir,[25] too, cited one of the most fulminating instances of acute isolated myocarditis found in an apparently healthy male who collapsed and died while shoveling snow. Another recent study [25] of isolated myocarditis in children showed that 3 out of 15 died while being admitted to the hospital.

Sudden death in diphtheritic myocarditis is common and well known but occurs infrequently when compared with rheumatic carditis. Griffith and Huntington [26] could find only 3 such cases of sudden death among over 7000 cases of active rheumatic carditis. Gold [27] recently reported the case of a child's sudden death due to rheumatic myocarditis and cited percentages of deaths caused by it from 2.5 to 9.8 per cent. Likewise, Lisa [28] stated that among 117 patients who had myocarditis, 59 patients died suddenly. In this same study emboli in branches of the coronary arteries were found in 5 of 15 hearts. The source of these emboli, according to Lisa, was most likely mural thrombi in the left ventricle, seemingly the result of the adjacent myocardial inflammatory changes. It was felt that a heart already handicapped by acute myocarditis might well become suddenly insufficient if additional impairment was brought about by small coronary artery emboli. Saphir,[25] too, listed other explanations for unexpected death of myocarditis; namely, sudden circulatory collapse or shock secondary to myocardial failure. The inflammatory exudate may involve the fibers of the conduction system or the nodal tissues producing ventricular fibrillation. A massive exudate with severe degeneration, necrosis or compression of many heart muscle fibers may exist; however, the prognosis, according to Saphir, does not depend so much on the number of muscle fibers which are incapacitated by the inflammatory process, but rather on the state and amount of uninvolved myocardium.

OTHER CARDIAC CAUSES OF SUDDEN DEATH

Sudden death in calcific aortic stenosis, according to Oram,[16] may result from fatal syncope or from angina pectoris which is present in 20 per cent of patients with aortic stenosis. Angina in such cases appears to depend upon poor coronary filling secondary to a jet effect or to a low mean aortic pressure. McGinn and White, Margolis and coworkers, Bergeron et. al., Contralto and Levine report sudden deaths in 5 to 15 per cent of their cases.[8] Of 76 patients studied by Willins,[8] 8 died unexpectedly or suddenly, the cardiac syncope of aortic stenosis being due probably to ventricular fibrillation. De Veer [16] considered that in some cases sudden death could be explained by the locking of a rigid, but free, aortic cusp against fused cusps in diastole. Coronary occlusion is the most common cause of sudden death in aortic stenosis; yet, there are probably multiple causes.

According to Grulee [29] congenital anomalies of the heart and great vessels are found in approximately 4 per cent of autopsies in hospitals caring for infants and children as compared to an incidence of

about 1 per cent in general hospitals. Grulee further pointed out that unexpected death in the neonatal period can occur from pulmonary artery atresia, complete transposition of the great vessels, persistence of primitive aortic arches as a vascular ring, anomalies of the coronary circulation, and coarctation of the aorta. Farber,[30] also, considered congenital anomalies of the cardiovascular system as one of the principle causes of sudden death in early life.

Sudden death has been observed in Ebstein's disease and idiopathic cardiac hypertrophy, too, according to Rosenbaum and Stiles,[31] who reported two cases of cyanotic congenital heart disease in whom an unusual mechanism occurred, thereby causing sudden death. In one patient the major lesions were pulmonary atresia and tricuspid stenosis, and in the other the chief anomalies were aortic and mitral stenosis. In both patients thrombi developed above the anomalous semilunar valves and ultimately became large enough to obstruct vital circulatory pathways and produce sudden unexpected death. These authors pointed out that, as surgical procedures are developed for alleviation of congenital anomalies, it is possible that care will have to be taken to avoid creation of cul de sacs in which thrombi form and lead later to serious consequences.

Cardiac catheterization fortunately is rarely a cause of sudden death; however, Goodwin [32] has described one case in which trauma to the endocardium of the outflow tract of the right ventricle resulted in unexpected death.

Sudden death may occur after cardiac trauma with or without preceding symptoms of angina pectoris. In an experiment conducted with dogs, it was found that a heavy blow may bruise the heart and cause ventricular fibrillation or even cardic rupture. Bright and Beck [35] feel that man probably reacts in a similar way.

The course and prognosis of tumors of the heart vary with the nature of the tumor, but sudden death is not uncommon even with benign tumors, such as myxoma or rhabdomyoma.

Kulka [33] pointed out that sarcoidosis of the heart caused sudden death; Stevens [34] recently reported a case with extensive myocardial sarcoidosis who died suddenly.

Another cause of unexpected sudden cardiac death is a ball valve thrombus which may completely occlude the mitral orifice.

THERAPEUTIC DRUGS

Of drugs used for treatment which can cause sudden death, digitalis, adrenalin, quinidine, and intravenous diuretics are undoubtedly the best examples. If given in sufficiently large doses in man, digitalis can induce any arrhythmia, including ventricular fibrillation. Likewise, adrenalin in large doses, especially if given intravenously by accident, will cause sudden death. In such cases almost any arrhythmia except heart block may be induced. Quinidine therapy also brings about unexpected death. In a review of 611 previously uncollected cases between 1947 and 1954, Thompson [36] noted that 20 deaths occurred during therapy and were variously interpreted by several authors. Clearly, in certain cases, death could be attributed to other causes, making the adjusted death rate 2.1 per cent which was regarded as the maximal risk of sudden death from quinidine in this series. It was apparent that the risk of sudden death from quinidine is real, and in some cases not susceptible to pathologic examination. No final conclusions regarding the cause of sudden death could be made because of the limitations of the reported autopsy material. However, the data favored some reason other than embolism. Severe organic heart disease, congestive heart failure, and associated serious illnesses increased the possibility of a fatal reaction. Thompson [36] stated that idiosyncrasies to the drug, the occurrence of ventricular tachycardia and fibrillation particularly associated with intravenous administration of quinidine and conduction defects, such as complete bundle branch block or intraventricular block, are known mechanisms of producing sudden cardiac death. Unappreciated toxic effects of the drug, especially on the central nervous system, also appear to play a role in the production of sudden death. It was suggested by Thompson [36] that these effects might be avoided by a more judicious se-

lection of patients for therapy and meticulous supervision of all patients receiving large doses of the drug.

CLINICAL ASPECTS AND TREATMENT

Whatever the cause of loss of consciousness, the clinical picture will be similar. The attack occurs at any time with dramatic suddeness and without warning. The patient loses consciousness immediately and at first is white, flaccid, pulseless and has no audible heart sounds although breathing may continue. The pupils are dilated. After approximately fifteen seconds, anoxic twitching begins and may proceed to a generalized convulsion. If the patient does not recover within three minutes, death usually results; but if recovery does follow, return to consciousness is abrupt and complete, and the patient's color changes from white to bright red because of hyperoxygenated blood being suddenly pumped into a dilated vascular system.

It seems paradoxical to discuss the treatment of sudden death due to heart disease, yet often the immediate cause of death is not irreversible, if treatment is instituted promptly.

Cardiac arrest is defined as the sudden stoppage of effective heart action due to cardiac standstill or to ventricular fibrillation. Stephenson [37] and associates, who collected over 1,200 such cases reported 90 per cent occuring as cardiac standstill and 10 per cent as ventricular fibrillation. Cardiac arrest may occur during any operation with any type of anesthesia; however, it does not occur exclusively in the operating room. Beck [38] described the case of a physician who "died" from an acute myocardial infarction while he was leaving the hospital. Cardiac massage was begun within three minutes after thoracotomy. Ventricular fibrillation was observed and a normal sinus rhythm was restored to the heart with an electric shock. The patient recovered. A similar case was reported by Regan [39] et al. Celio,[40] too, successfully resuscitated a patient with an acute anteroseptal myocardial infarction who suddenly developed cardiac arrest. Recently, Hannon [41] reported a case in which a hospital employee stricken in the hospital corridor was resuscitated from a cardiac arrest secondary to myocardial infarction.

Treatment of cardiac arrest requires prompt recognition, previously organized preparation with a predetermined plan of action, and readily available equipment. A so-called cardiac resuscitation kit which includes a defibrillator should be available at critical locations in every hospital.

Because of the absence of bleeding and the permissibility in such cases of ignoring the danger of infection, cardiac massage can be performed by doctors with no surgical training. This procedure should be studied and adopted by internists and general practitioners as well as by surgeons. Once cardiac massage has been instituted, the anesthetist can be summoned to supervise controlled respiration; the surgeon, to attend the wound. If there is to be successful resuscitation, it must be undertaken immediately, as delay incurred in sending for a surgeon and an anesthetist involves the danger of irreparable cerebral damage.

Another means of treating cardiac standstill is by the use of the external electric pacemaker. Zoll [42] and associates successfully resuscitated 27 patients from Stokes Adams attacks with the electric pacemaker. Rosenfeld and Segall [43] reported 3 cases and Kaye,[44] 1 patient with Stokes Adams disease, all of whom were revived with the external electric pacemaker. The external pacemaker was repeatedly effective in resuscitating three patients with ventricular standstill due to reflex vagal stimulation.[42] The precipitation of such episodes by carotid sinus pressure and their disappearance after the administration of atropine established their reflex vagal origin. External electric stimulation terminated ventricular standstill and restored an effective circulation in these patients more than one hundred times. Promptly resuscitated with the pacemaker by Zoll and associates were 2 patients who had ventricular standstill produced by digitalis intoxication. Besides, 2 patients with ventricular standstill after intravenous administration of procainamide in the treatment of ventricular extrasystoles and tachycardia were also resus-

citated immediately by the external pacemaker.

It may well be concluded that the external electric pacemaker is a convenient and practical means of reanimating the arrested heart. The ultimate prognosis depends on the underlying etiologic factors disturbing the conducting mechanism.

SUMMARY

1. Sudden unexpected death due to heart disease occurs frequently.

2. The clinical causes and treatment of sudden cardiac death have been discussed.

3. Every hospital should have the necessary equipment available for the treatment of cardiac arrest.

4. Cardiac arrest does not occur exclusively in the operating room, but occasionally follows coronary occlusion, Stokes Adams attacks, reflex vagal stimulation and drug intoxication.

5. Some cardiac cases can be resuscitated, since death is often due to a disturbance of rhythm rather than myocardial failure.

6. Internists and general practitioners should be prepared to resuscitate with or without the external electric pacemaker the instant cardiac arrest occurs.

7. An active interest in therapy for cardiac arrest will salvage some patients from sudden cardiac death.

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MYOCARDIAL INFARCTION—EMERGENCY TREATMENT *

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From continual improvement in diagnostic methods, we have become more aware of the wide variations possible in the clinical picture of acute myocardial infarction; it follows that there can be no set routine emergency treatment covering all cases with this condition. Differences of opinion exist in the management of certain phases of myocardial infarction, yet the basic therapeutic principles have undergone little or no change in the past several years. The purpose of this paper is to discuss these therapeutic principles, and to state and discuss my personal opinion regarding the management of the emergency phase of this condition. This outline is based, primarily on my clinical impressions gained through personal experience in the field of cardiology. Though the exact limits of the emergency phase of acute myocardial infarction may defy definition, I will consider the first seventy-two hour period as the emergency phase. This will allow us a somewhat general agreement of terms.

In management of acute myocardial infarction, the physician must evaluate each patient individually. This evaluation must be made in respect to the presenting clinical signs and symptoms as well as the personality of the patient and his reaction to the disturbance. Particularly in the emergency phase, the treatment should be directed at the patient and not the magnitude of the QRST changes on the electrocardiogram. Treat the patient—not his electrocardiogram.

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HOSPITALIZATION

It is my opinion that all patients with acute or impending infarction should, if at all possible, be hospitalized regardless of the severity of the presenting symptoms or the electrocardiographic changes. Routine hospitalization is indicated by the constant uncertainty of these cases, especially in the first few days. It is true that many patients with so-called "mild attacks" have been and are still being successfully treated at home, but this does not per se support an argument for home treatment. All too often, I have viewed "mild" cases, being treated at home, to exhibit a recurrence of chest pain, go into shock, and die before any emergency treatment can be initiated. It is evident that hospitalization makes certain necessary treatments more readily available; that is, use of opiates, vasopressor drugs and oxygen therapy. In addition, hospitalization affords the physician the opportunity to observe his patient more carefully so as to detect and treat impending complications and disturbances; and to more accurately evaluate the patient's progress in order to make recommendations regarding future activity and his ultimate return to work.

When and how a patient should be moved to the hospital is a frequent problem. Mild cases in which the presenting signs and symptoms have subsided may be safely transferred by automobile. Most other cases should be transported by ambulance. Severe cases in which intractable pain is exhibited should not be moved until pain has subsided or disappeared. Patients presenting the picture of shock should receive vasopressor drugs before transfer to the hospital.

CONTROL OF PAIN AND ANXIETY

The control of pain and anxiety is the

first and most common problem with which the physician is confronted in a case of acute myocardial infarction. The treatment of these symptoms and choice of drug should be so designed as to give the patient maximum relief without producing serious or unpleasant side effects.

In certain instances in which pain has subsided, sedation may be all that is necessary. For patients experiencing mild pain, dolophine®, demerol®, or codeine will usually be adequate. Nitroglycerine does not, as a rule, relieve the pain of acute myocardial infarction; however, it may afford temporary relief in certain mild cases. This drug should be used cautiously in patients having severe pain with impending shock, for it frequently causes a further drop in blood pressure, thereby decreasing, instead of increasing, the coronary blood flow.

Morphine, although it frequently produces undesirable side effects, in my experience, is still the drug of choice not only because of its pain relieving powers, but also because of the euphoria and sedation it produces. The average case requires from 1/6 to 1/4 grain hypodermically for relief. If a more rapid effect is desired, the drug should be given intramuscularly and the injection area massaged. For intractable pain this dose may be repeated in thirty minutes to an hour. As much as 1 grain may be required in severe cases. Occasionally, it may be necessary to give from ¼ to ½ grain of morphine intravenously for relief of extremely intense pain. For this route of administration, the drug should be diluted with 10 cc. of sterile water or saline and given slowly until the pain subsides. Dilaudid®, 1/32 grain, is as effective an analgesic as morphine, and it is preferred by some physicians on the basis that it produces less nausea and vomiting. Demerol®, 75 to 100 mg., hypodermically, may suffice to control pain. Although not possessing the pain-relieving qualities of morphine, demerol® does not cause respiratory depression to the degree of morphine and the other opiates. I very seldom use it initially, as I have found that its relaxing effect on smooth muscle not infrequently causes a drop in blood pressure, especially in patients with infarction accompanied by signs of impending shock. This induced state of shock, precipitated by demerol®, is more likely to occur when this drug is used in combination with aminophyllin, another potent vasodilator. Dolophine®, 5 to 10 mg., is another effective analgesic and very seldom causes nausea and vomiting. I have found it very useful, in combination with phenergan®, for patients that cannot tolerate morphine or dilaudid®. Oxygen, aminophyllin, and papaverine have been recommended for intractable pain, but I have not been impressed with their efficacy in this regard.

Because opiates have a depressing effect on the respiratory center and may induce Cheyne-Stokes respiration, large doses of these drugs should be used cautiously. Not infrequently, the average dose of morphine will produce marked respiratory depression, particularly in a debilitated and elderly patient with cerebral arteriosclerosis. When excessive narcosis occurs, or the respiratory rate falls below 12 per minute due to opiates, Nalline®, 10 mg., should be given intravenously and repeated in ten to fifteen minutes if necessary. If an increase in respiratory rate is not evident in two to three minutes, an additional injection of 10 mg. may be given. Aminophyllin, 10 cc. (3 and ¾ gr.) intravenously, is the drug of choice for Cheyne-Stokes respiration. Occasionally, atelectasis and/or pneumonia will develop following the use of large doses of opiates during the initial period of treatment. This is due to depression of the cough reflex and retention of secretions which are normally expectorated. To prevent such a complication, the patient should be encouraged to take deep breaths frequently, and to cough if there are any indications of secretions present in the bronchial tree.

SEDATIVES

Sedatives, as a rule, are not necessary early in the treatment of myocardial infarction when morphine or other opiates are used. Patients having transient pain due to impending infarction should be given a sufficient amount of sedation to produce mild drowsiness. For this purpose, phenobarbital, in ½ gr. doses t.i.d., is still the

drug of choice. I have not found that meprobamate or the other tranquilizers offers the patient any more advantages than the barbiturates. One should remain aware, of course, of the fact that in some patients the barbiturates will induce excitement and restlessness instead of the desired depression. Chloral hydrate, 7-½ grains, is an effective sedative and may be substituted when patients are sensitive to barbiturates. In my experience, doriden®, 0.5 gm., has proved to be an ideal drug for nocturnal sedation.

BED REST

Complete bed rest has been traditionally regarded as the most effective means of affording the injured heart maximal rest. Until a few years ago, patients with acute myocardial infarction were placed in the recumbent position and not allowed to move an extremity or turn around in bed unassisted. The trend lately has been towards a liberalization of the rigid restrictions of complete bed rest, allowing the patient to move more freely in bed. Physiological data presented by many recent writers show that the cardiac output is significantly less in the sitting position than in the recumbent position. In the past year I have encouraged my patients from the very beginning, to spend as much time as possible in the sitting and semirecumbent positions. I have found that these patients often get along better, recuperate physically and psychologically much faster, and develop fewer bowel and bladder complications. For the first few hours when the patient is having acute symptoms, the position of the bed should be adjusted to his comfort.

Those cases not complicated by signs of peripheral vascular collapse are placed in the sitting position for meals and are allowed to feed themselves if this slight exertion does not initiate chest pain. They are encouraged to remain in the sitting position until fatigue develops, and then the head of the bed is lowered to the semirecumbent position. Patients that have difficulty using the bed pan are allowed to use the bedside commode. Cases dominated by severe and prolonged pain are treated in the same manner except that they are not allowed to feed themselves.

When the picture is complicated by shock, the patient is placed in the recumbent position until the blood pressure is stable and all signs of peripheral collapse have disappeared. In the presence of left-sided heart failure, the patients are usually more comfortable in the sitting position. All patients are encouraged to take deep breathing exercises, dorsiflex both feet. and contract leg muscles frequently.

ANTICOAGULANTS

Though the use of anticoagulants in the treatment of acute myocardial infarction is now generally accepted, there still exist some differences of opinion as to which cases should receive these drugs. Recent writers on the subject strongly favor the use of anticoagulants for all cases of myocardial infarction regardless of the severity of the attack or the presence, or absence, of other specific indications for their use. With few exceptions, most of the currently reported series show that there is a statistically significant decrease in the mortality rate as well as a decrease of the incidence of thromboembolic complications in those patients treated with anticoagulant drugs.

There are some authors who still maintain that anticoagulants are neither necessary nor desirable in every case of acute myocardial infarction. These authors divide their patients into two general groups of "good-risk" patients and "poor-risk" patients. The "good-risk" patients, those having a first attack without serious prognostic signs, are not given anticoagulants on the basis that the mortality rate is low and the use of these drugs would not favorably influence the outcome of these cases. The popular argument against this premise is that one cannot accurately predict during the first few hours or days, which case will be mild or severe; and since it is presumed that thromboembolic complications are more likely to occur in the first few days after the acute episode, the prompt institution of anticoagulant therapy is thought desirable.

It has been my practice in recent months,

to place all patients on anticoagulants immediately after the onset of the acute symptoms, whenever adequate and competent facilities are available for continued determination of coagulation and prothrombin times. Patients who, in my estimation, have a "mild" attack and have not known predisposing thromboembolic factors are usually maintained on anticoagulants until fully ambulatory, i.e., about six weeks. "Severe" cases are now being maintained on anticoagulants for several months or more, depending on the presence of factors known to favor the development of thrombi. Those patients who develop symptoms of coronary insufficiency or impending infarction after the first attack are usually maintained on therapy for several months after symptoms have subsided. Though the previous statments are not concerned solely with the emergency treatment of myocardial infarction, I have included them here for I feel that any current discussion of the subject demands at least this degree of comment on anticoagulants.

Initially, the combination of heparin and dicumarol or other cumarin derivates should be used. Several methods of administering heparin are available, but I have found that giving 50 mg. doses subcutaneously every six hours for thirty-six to forty-eight hours to be the most practical regimen. At the same time heparin is started, the patient is given a 300 mg. dose of dicumarol. A prothrombin determination is performed on admission and repeated daily while the patient is in the hospital. Subsequent dicumarol dosages are then adjusted according to the prothrombin time. For clinical purposes, two to two and one-half times the control prothrombin time is considered the accepted therapeutic range. Heparin is discontinued when the prolongation of the prothrombin time is within these limits. Even though the development of a hemopericardium is not a common complication of anticoagulant therapy, it has been my custom to discontinue these drugs temporarily on all cases that evolve a significant pericardial friction rub.

Anticoagulants are contraindicated in the presence of prolonged prothrombin time due to Vitamin K deficiency or severe liver disease, renal insufficiency, acute gastric or duodenal ulcer, blood dyscrasias with impairment of normal mechanism of hemostasis, and/or recent surgical operation or open wounds.

OXYGEN

Oxygen has been widely used in the emergency treatment of acute myocardial infarction; however, doubt exists as to its true need in all such cases. Studies show that the arterial oxygen saturation is essentially normal in the uncomplicated cases, yet those patients with evidence of shock and/or pulmonary edema may exhibit a significant degree of anoxia. I administer oxygen to all such complicated cases as well as to those patients with intractable pain.

Oxygen may be administered by nasal catheter, mask, or by tent. The recommended method would depend on the severity of the anoxia and the comfort and wishes of the patient. Recently, the administration of oxygen by intermittent positive pressure has become more popular in the treatment of pulmonary edema.

SHOCK

Shock is a serious and often fatal complication of acute myocardial infarction and, therefore, should be watched for and treated immediately. The resulting mortality rate is high and is directly proportional to the severity and duration of the shock state. Since the advent of effective vasopressor drugs, the mortality rate is significantly reduced. Clinical signs of shock almost always develop soon after the onset of acute infarction symptoms and persist from a few hours to one or two days. Transitory fall of blood pressure is a common finding in myocardial infarction of moderate or mild degree and does not require specific treatment. Whenever the systolic pressure falls below 90 mm. of Hg. in a normotensive patient and below 100 mm. of Hg. in a hypertensive patient, very careful observation is imperative. Quite frequently, patients in mild shock will improve with only bed rest, oxygen, and alleviation of pain and anxiety with opiates. Those cases that do not respond to the usual treatment for acute myocardial infarction

should be given one of the vasopressor drugs.

My practice is to administer oxygen to all patients in shock. Those presenting the picture of "mild" shock are given 15 to 30 mg. of wyamine® (mephentermine). intravenously. Further drug therapy is withheld if the systolic pressure is maintained at or above 100 mm. of Hg. When wyamine® is ineffective in relieving shock, an intravenous infusion of 5 per cent dextrose in distilled water which contains from 4 to 12 mg. of levophed® (norepinephrine) is instituted immediately. Patients with acute infarction initially exhibiting profound shock should be instantly started on a levophed® drip. The infusion flow is regulated so as to maintain a systolic pressure of 100 to 110 mm. of Hg; usually 20 to 30 drops per minute is sufficient. If the desired pressor effect is not obtained, more levophed® is added to the infusion bottle—this being preferred to an increase of the infusion drip rate. When the blood pressure has been maintained for several hours, the levophed® is gradually reduced and eventually stopped if the blood pressure level is adequate and stable. During administration of vasopressor drugs, the constant attendance of physician or nurse is mandatory for the continual check on blood pressure effects. Those patients who indicate little ability to sustain their own pressure after a reasonable period of time (six to eight hours), are given corticotropin hormone (ACTH) either intravenously or intramuscularly. In many instances, the response is dramatic and the levophed® may be discontinued within a few hours.

CONGESTIVE HEART FAILURE

Some cases of acute myocardial infarction will be ushered in with intense dyspnea, anxiety, and other signs of pulmonary edema. Because symptoms usually appear suddenly and require immediate attention, treatment, in most cases, must be initiated in the home. Morphine or other opiates will frequently afford rapid and dramatic symptomatic relief. In the presence of bronchospasm, aminophyllin gr. 3-¾, is often very effective. If improvement does not occur rapidly with these measures, the patient should be digitalized with a fast acting digitalis preparation such as, ouabain, lanatoside C, or digoxin. At the same time 2 cc. of a mercurial diuretic should be given intramuscularly. As soon as possible, oxygen should be administered in high concentration or under positive pressure. If there are signs of rightsided heart failure, as evidenced by distended neck veins, the removal of 350 cc. to 500 cc. of blood may be lifesaving.

ARRHYTHMIAS

Almost any of the cardiac arrhythmias may develop during the early days following acute infarction. Premature ventricular contractions are the most common but rarely require treatment. It is my custom to prescribe quinidine or pronestyl® only when the contractions are disturbing to the patients or recur frequently. Quinidine in doses of 3 to 6 grains or pronestyl® in doses of 250 to 500 mg., three to four times daily, is usually adequate to control this abnormality. Because these drugs not infrequently cause a significant drop in blood pressure, they should be used cautiously in the first few days following infarction. Auricular fibrillation is usually a transient disturbance which often subsides spontaneously with a little sedation. When it persists and the ventricular rate is rapid, immediate digitalization to control the apical rate between 70 to 80 beats per minute is indicated. Quinidine is used with digitalis only when frequent ventricular premature beats are present before digitalization, or develop shortly thereafter. Auricular flutter is treated in the same manner. Ventricular tachycardia, as a complication of acute infarction, indicates an extremely grave situation and must be treated immediately. Quinidine and pronestyl® are the drugs of choice and, in my opinion, are equally effective for this particular arrhythmia. Some cases may be managed with oral medication, but because of the seriousness of the condition, the parenteral route of administration should be used in most instances. Quinidine, when given orally, is usually started at a dose of 3 grains every two hours. If the arrythmia does not respond, the dosage is increased to 6 grains

every two to three hours. When the parenteral route is indicated, quinidine gluconate is given in 0.6 to 0.8 gm. doses in 250 cc. of 5 per cent dextrose administered over a period of one hour under electrocardiographic control. Pronestyl®, in doses of 200 to 1000 mg., may be given intravenously, at a rate not to exceed 100 mg. per minute. The blood pressure should be recorded frequently and if a significant hypotension develops, wyamine® or levophed® should be administered immediately. Supraventricular tachycardia may be terminated by sedation and/or carotid sinus stimulation, but quite often rapid digitalization is necessary.

REASSURANCE

Though reassuring the patient who has suffered a myocardial infarction is extremely important, it is not necessarily easy; for most people today are aware of the seriousness and uncertainties of the condition and generally familiar with the effect it has on one's future. It is comforting to the patient to know what to expect; hence from my experience, a careful discussion, in simple language, of the condition and symptoms is recommended. Such a discussion, in which it is pointed out that the majority of cases recover completely and resume normal activities, I have found, is a most effective method of controlling the profound psychological disturbances that almost invariably develop. Explaining what has gone wrong and what you hope to accomplish with your therapy helps the patient to feel that he is in competent hands and that everything possible for his comfort and recovery is being done. I also find it helpful to explain to the patient that certain symptoms, like pain, do not in themselves indicate a bad prognosis. When particular physical findings are normal, I feel that the patient benefits from being informed. If his pulse, blood pressure, and/or heart sounds are normal, I like to tell the patient so; and that he is doing fine. The beneficial results of the physician's display of confidence in himself and his knowledge have been often observed. On many occasions, the cardiologist is called in consultation not because the case is a problem in diagnosis or management but purely for the beneficial psychological effect.

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DRUG THERAPY OF CARDIAC ARRHYTHMIA *

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Some arrhythmias, such as sinus arrythmias, sinus bradycardia, and some cases of complete heart block, require no treatment. In others, some aspects of treatment are still controversial. I will try to present the latest advances in treatment and the type of treatment most universally accepted.

PREMATURE SYSTOLES

Actually they are of little clinical significance in most cases; however, due to their common occurrence and the danger of overemphasizing their importance to

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the patient, I will mention them. Premature beats are due to abnormal stimuli arising in some area of the heart outside the normal pacemaker and originating a beat before the regular beat.

If the premature beats are very frequent or disturb the patient, treatment is indicated. Any possible causative agent such as tobacco, alcohol, fatigue, or disease should be eliminated if possible. If there is no associated heart disease, mild sedation is effective. Quinidine, in 0.2 grams to 0.4 grams, four times a day will usually suppress the prematures. If they are due to digitalis intoxication, potassium salts will control them. When there is evidence of congestive failure, digitalis, by improving the heart function may control the prematures.

SUPRAVENTRICULAR TACHYCARDIAS

Paroxysmal supraventricular tachycardia is a term used to indicate auricular or atrioventricular nodal tachycardia. The treatment of both is similar and they are frequently difficult or impossible to differentiate. Paroxysmal auricular tachycardia is by far the more common of the two. Usually this fast rhythm, especially when auricular in origin, is very regular and has a sudden onset and cessation. Its usual rate is between 160 and 220. These arrhythmias are often imposed on otherwise normal hearts.

In the treatment of these tachycardias, an attempt should first be made to eliminate any possible causal agents such as tobacco, fatigue, emotional upset, infection, digestive disturbance, thyrotoxicosis, and digitalis intoxication.

Stimulation of the vagus slows the sinoauricular node rate, slows conduction through the atrioventricular node and depresses any ectopic focus in the auricles. The simplest and quickest treatment is vagus stimulations by carotid sinus pressure, eyeball pressure, Valsalva maneuver, or induction of vomiting.

The carotid sinus is located at the bifurcation of the carotid artery at the angle of the jaw. Pressure should be applied firmly toward the vertebral column for ten to fifteen seconds in a rotary manner at the bifurcation of the carotid. Pressure applied proximal to the bifurcation will result in lower intraluminal pressure in the sinus and actually cause vagal inhibition. This mistake is a frequent cause for failure of this maneuver. The right side is more sensitive usually and should be pressed first. If either carotid has decreased pulsation, pressure should be applied for a shorter period of time. Massage of both carotids at the same time markedly reduces cerebral blood flow and should not be tried except in young people under special circumstances.

On eyeball pressure increased stimulation of the vagus is transmitted through the ophthalmic branch of the trigeminal nerve to the vagal nuclei. Pressure should be applied on both eyes to the point of pain for twenty to thirty seconds. The danger of retinal detachment during this procedure has been mentioned but I think it is remote.

In the Valsalva maneuver, the patient maintains forced expiration with the glottis closed for fifteen to twenty seconds. The postexpiration rise in blood pressure will increase pressure in the carotid sinus and thus cause vagal stimulation.

Vomiting can be caused by mechanical irritation of the pharynx or by ipecac in rather large doses, four to eight cc. initially. This dose can be repeated in forty-five minutes if normal sinus rhythm has not returned. Apomorphine is not as safe or effective as the above two means.

Sedatives and narcotics, especially morphine, relieve anxiety and emotional tension and, when no heart disease is present, will frequently relieve the tachycardia. However, the use of narcotics is always dangerous in any recurring illness.

If the above simple measures fail and the patient is not on digitalis, this drug is indicated. Of course, digitalis is especially beneficial when failure is present. One of the rapidly acting digitalis preparations such as lanatoside C (Cedilanid) is preferable. Depending on the urgency of the situation they may be given either intravenously or intramuscularly.

A sound approach is to give 0.8 milligrams of Cedilandid intravenously or intramuscularly and repeat in one hour if necessary. Since digitalis increases vagal tone, some of the maneuvers such as carotid sinus pressure should be used in conjunction with digitalis. Before digitalis is used, ventricular tachycardia must be ruled out.

If the supraventricular tachycardia is thought to be due to digitalis intoxication, this drug should be discontinued and the patients with normal kidney function should be treated with potassium salts, either orally or intravenously.[1] Twenty milliequivalents of potassium diluted in 200 to 300 cc. of 5 per cent glucose in water can be given intravenously. The patient should be followed closely by electrocardiography during the infusion. This dose may be repeated or given orally as 2 grams of potassium chloride every four hours.

The uses of quinidine, as outlined in the discussion of auricular fibrillation, is applicable in supraventricular tachycardia and will not be discussed here.

Neo-Synephrine intravenously causes a rapid rise in aortic and carotid sinus pressure which in turn increases vagal tone. A number of writers have commented on the speed and effectiveness of this treatment.[2] The initial dose is 0.5 milligrams intravenously. This dose may be increased by 0.5 milligrams each time until normal rhythm is established and repeated as soon as the blood pressure has returned to pre-injection levels. Hypertension and coronary disease are frequently mentioned as relative but not absolute contraindications. Donegan and Townsend[3] point out that Neo-Synephrine does occasionally cause enough myocardial irritability to produce short runs of ventricular tachycardia and is potentially dangerous. They recommend the use of this drug after the safer therapies have been tried.

McGinn and Schluger[4] point out that the same results can be obtained with levarterenol bitartrate (Levophed) given intravenously and recommend its use in all attacks in which the blood pressure is low or normal. Occasional paroxysms of ventricular tachycardia are caused by Levophed and a solution of procaine amide should be available for immediate intravenous administration. Levophed should be given at a rate to raise the blood pressure quickly to 120 to 160 millimeters of mercury.

Parasympathetic drugs increase vagal tone and are beneficial in treatment of supraventricular tachycardia. The side effect such as salivation, nausea, muscle twitching, etc. can be controlled by atropine in doses of 0.5 to 1.0 milligrams intravenously.

Mecholyl can be given initially in doses of 5 to 15 milligrams subcutaneously. A larger dose may be given after twenty minutes. The average dose is about 25 milligrams. Usually the drug is effective in five to ten minutes. Acetylcholine intravenously in an initial dose of 20 milligrams will frequently relieve the tachycardia and has considerable less side effects than mecholyl. It can be repeated every three or four minutes in successively increasing doses (40, 60, 80, and 100 milligrams). Neostigmine given subcutaneously, intramuscularly and intravenously, in doses of 0.5 to 1.0 milligrams, is frequently effective and apparently quite safe.[1] If these parasympathetic drugs do not end the attack, they should be supplemented by carotid sinus pressure.

In summary, supraventricular tachycardia can be treated first by sedation and the various maneuvers to increase the cardio-inhibitory reflexes. If these fail, Cedilanid should be used to be followed by Neo-synephrine or one of the cholinergic drugs provided the patient is known not to have too distressing side effects to them. In conjunction with those three drugs, carotid sinus pressure should be used. If the above fail, a thorough trial of quinidine is in order.

ATRIAL FLUTTER AND FIBRILLATION

Prinzmetal[5] has produced strong evidence that auricular flutter and fibrillation are not due to the circular movement as described by Lewis. He has shown

that auricular flutter can be caused by rapid discharges from an ectopic focus in the atrium. Flutter changes into fibrillation when the rate of discharge from the ectopic focus increases above a critical level called by Printzmetal the fibrillation threshhold. At this level,[6] there is a complete breakdown of orderly electrical and mechanical activity.

In the treatment of the auricular flutter and fibrillation, any known etiological factors such as thyrotoxicosis should be eliminated. The drug of choice for flutter or fibrillation is digitalis. Full digitalizing doses are required to reduce the ventricular rate.

In flutter after full digitalization, the patient may convert to sinus rhythm, the flutter may persist with the same or more A-V block, or atrial fibrillation may develop. Some cardiologists recommend discontinuing the digitalis if fibrillation develops in the hope that a normal sinus rhythm will be produced. However, I believe if flutter persists or fibrillation develops, digitalis should be continued in maintenance doses and quinidine therapy added.

The use of quinidine for digitalized cases of flutter and fibrillation is similar and will be discussed together. Auricular fibrillation is an uneconomic cardiac rhythm and with few exceptions conversion should be attempted with quinidine. Cases[7] in which conversion should not be attempted are (a) cases with complete heart block, (b) elderly patients with old fibrillation whose failure is well controlled by digitalis and a low salt diet and who do not have a history of embolic phenomena. Also, patients with a history of severe sensitivity reaction to quinidine such as thrombocytopenic purpura and fever, should not be treated. Before quinidine therapy is given, most experts feel that the fibrillating patient should be on anticoagulant therapy for ten to fourteen days. This is to prevent any recent thrombosis formation that might be the source of emboli during conversion to sinus rhythm. However, a number of authors[8, 9, 10] point out the rarity of embolization during conversion and question whether the benefits from anticoagulants outweigh their possible dangers and the added time and expense necessitated by them.

Bedard[9] reports therapy had to be stopped in 6 cases out of 67 treated with quinidine due to toxic complications or side effects. Severe gastrointestinal upset, frequent ventricular ectopic beats, ventricular tachycardia, transient ventricular fibrillation, and nodal rhythm are indications for discontinuing treatment. Prolongation of QRS intervals over 50 per cent[12] and increasing degrees of heart block also require discontinuance of treatment. High fever or blood dyscrasias in rare instances may force abandonment of therapy. Patients should have an electrocardiogram and should be seen by a physician before the daily course of quinidine therapy.

Most authorities[7, 9] recommend starting quinidine therapy, after an initial test dose, on a schedule of 0.2 grams every two hours for five doses making a total dose of 1.0 gram the first day. This dose is increased 0.1 gram per dose daily, that is, 0.3 grams five times the second day, etc. (Table 1). This schedule may be continued until 1.0 gram doses are being given, which amounts to 5.0 grams per day.[11] When more rapidly increasing action is indicated the dose may be increased 0.1 grams to 0.2 grams after three or four doses and this schedule repeated until normal rhythm develops or toxicity occurs.

TABLE 1.
QUINIDINE DOSE SCHEDULE

Day	Individual Dose	Total Dose
1.	0.2 grams q 2 hours x 5	1.0 grams
2.	0.3 grams q 2 hours x 5	1.5 grams
3.	0.4 grams q 2 hours x 5	2.0 grams
4.	0.5 grams q 2 hours x 5	2.5 grams
5.	0.6 grams q 2 hours x 5	3.0 grams
6.	0.7 grams q 2 hours x 5	3.5 grams
7.	0.8 grams q 2 hours x 5	4.0 grams
8.	0.9 grams q 2 hours x 5	4.5 grams
9.	1.0 grams q 2 hours x 5	5.0 grams

Sokolow[12] has pointed out the variations of blood levels in different people

on the same dose of quinidine. This variation in serum concentration on identical dose schedules may explain some of the failures of quinidine therapy. Maximum serum concentrations come about two hours after the last dose. After five or six doses every two hours, excretion and metabolic breakdown about balance the absorption and no further rise in level will occur unless the dose is increased.

If the drug is given every four hours, this balance is not reached until forty-eight to seventy-two hours. Some authorities recommend giving the drug every four hours in doses beginning at 0.2 grams and increasing every seventy-two hours by 0.2 grams per dose until the fibrillation is controlled.

In 700 cases [12] Sokolow found that the signs of myocardial toxicity were rare with serum levels of 6 micrograms or under. In a large series of patients 80 per cent of possible conversions were made with minimum toxicity on the dose of 0.6 grams every two hours for five doses or 3.0 grams per day. Higher doses gave only 20 per cent additional conversions and much toxicity.

Relapses usually are related to the low serum concentrations. Doses of 0.2 grams to 0.4 grams three times a day often do not give serum levels adequate to prevent recurring fibrillation. Frequently, doses of 0.4, 0.6, or even 0.8 grams every six hours are required.

Parenteral quinidine may occasionally be indicated. Quinidine gluconate is a very satisfactory preparation. The usual dose is 0.8 grams, intramuscularly, which may be given three times or rarely four times at one or two hour intervals. The peak serum concentration occurs about one hour after injection and rises rapidly on repeated injections. Occasional moderate hypotension results after intramuscular quinidine. Only under the most urgent circumstance is intravenous quinidine indicated. It can be given by diluting 0.8 grams of quinidine gluconate to 50 cc. and giving at a rate not to exceed 1 cc. or at most, 2 cc. per minute. An electrocardiogram should be taken constantly during the infusion and blood pressure watched closely. Rather marked hypotension occurs frequently.

I will not discuss the use of procaine amide (Pronestyl) in the treatment of auricular fibrillation. Its use as discussed under ventricular tachycardia will also apply to auricular fibrillation. It appears to be of some value in the treatment of auricular fibrillation of short duration.[1, 13]

VENTRICULAR TACHYCARDIA

Therapy in this arrhythmia is aimed at reducing the excitability or irritability of the ectopic focus producing the tachycardia. The drugs of choice are quinidine and procaine amide (Pronestyl). The use of quinidine has been discussed in connection with auricular fibrillation. Procaine amide can be administered orally, intramuscularly, or intravenously. Intravenously, the drug frequently produces rather marked hypotension and is rarely indicated as the drug is rapidly effective intramuscularly. When injected intravenously, it is best to dilute 1.0 grams in 100 cc. of glucose and give slowly at, not over, 100 milligrams per minute [13] until the desired effect is obtained or 1.0 to 2.5 grams [14] are given. The blood pressure should be followed closely and an electrocardiogram taken continuously. Due to the frequent hypotension produced, it is advisable to have a vasopressor drug available.

After intramuscular procaine amide (Pronestyl), an effective blood level is obtained in fifteen minutes to one hour. Hypotension is much rarer than with the intravenous route. The initial dose should be about 1.0 gram. If indicated, the subsequent dose of 1.0 gram can be repeated every one to three hours.

By mouth, the dose is usually 1.25 grams followed in one hour by an additional 0.75 grams.[13] If this dose is not effective, doses [1] of 0.5 grams to 1.0 grams repeated every two hours may restore normal rhythm.

Significant toxic effects have been limited to the gastrointestinal tract and circulatory systems. High oral doses (6 to

8 grams a day) frequently produce anorexia, nausea, and vomiting. Hypotension is infrequent on oral doses. A single oral dose of Pronestyl will give some transient electrocardiographic changes in about one-third of the patients that may persist on continuous therapy. These consist of widening of the QRS complex and ST interval, and lowering the amplitude of QRS and T waves. The prolongation of the QRS over 25 per cent is probably indication for discontinuing the drug. If the drug is given in large doses, the patient should be followed rather closely with electrocardiograms.

Of course, if ventricular tachycardia is due to digitalis intoxication, the treatment of choice is discontinuance of digitalis plus potassium therapy as outlined earlier.

STOKES-ADAM SYNDROME

Complete heart block with idioventricular rhythm is frequently associated with syncopal attacks. The majority of syncopal attacks [1] are due to ventricular asystole; however, they are sometimes due to ventricular tachycardia and fibrillation. If possible, the exact mechanism of the attack should be determined and the treatment based on this information. Of course, in well established complete heart block without symptoms, no treatment is necessary.

In the past, the treatment [10] of choice for these attacks has been epinephrine subcutaneously or intramuscularly. The main objection to epinephrine is its tendency to produce ventricular fibrillation.

Isopropylarterenol (Isuprel) has been reported as an effective drug in treatment of these attacks by several authors.[8, 10, 15] Isuprel causes an increase in cardiac rate and the amplitude of contraction. It increases cardiac irritability but has, to little if any degree, the undesirable tendency to produce ventricular fibrillation. Schumaker and Schmock [15] cite several cases in which ventricular tachycardia with complete heart block was effectively corrected by isuprel given subcutaneously. When the underlying mechanism of syncope is unknown, the treatment of choice is isuprel. The usual dose [1] is 0.2 milligrams subcutaneously. In very urgent cases, 0.02 milligrams may be given intravenously. For long term prophylactic therapy, the drug can be given sublingually in 15 milligram tablets three or four times daily.

It is important to emphasize here that procaine amide and quinidine are contraindicated in treating syncopal attacks associated with complete atrioventricular block.

Bellet and Brody [16] recommend molar sodium lactate intravenously for the treatment of syncope associated with asystole in complete heart block. Of course, the lactate must be given as soon as possible, preferably within one or two minutes after the attack has begun. The dose is 20 to 80 cc. given by syringe in a period of one to two minutes. Following this injection, an infusion should be started and the speed and the amount of molar sodium lactate regulated closely. When therapy needs to be given over a period of six to twelve hours, a rate of 15 to 20 drops per minute is usually satisfactory. However, if seizures tend to recur, the infusion can be speeded up.

The oral administration of molar sodium lactate as maintenance treatment to prevent frequent recurrence of the Stokes-Adams attacks is being investigated by several workers but I have no definite information about their conclusion except that the initial results do look encouraging. Molar sodium lactate is relatively nontoxic; the main toxic manifestation being the occasional occurrence of extra systoles and rarely pulmonary edema. Neither of these was any significant problem in the 46 cases in which molar sodium lactate was used by Bellet and Brody.

Beneficial effects of molar sodium lactate are probably due to the mild alkalosis it produces and to the rapid utilization of the lactate by the heart muscle as fuel.

SUMMARY

This paper reviews the drug therapy of premature systoles, supraventricular tachycardia, auricular flutter, auricular fibrillation, ventricular tachycardia, and complete heart block with Stokes-Adams

syndrome. The routes of administration and the dose of the various drugs are given.

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THE PICKWICKIAN SYNDROME *
A Case Report

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The association of extreme obesity, cyanosis, and somnolence was noted by Charles Dickens in the Pickwick Papers [1] in 1837, and was labeled "A Pickwickian Syndrome" by Burwell et al [2] in 1955. In the latter article, the syndrome in its entirety was described for the first time, and eight clinical features were listed: (1) marked obesity, (2) somnolence, (3) twitching, (4) cyanosis, (5) periodic respiration, (6) secondary polycythemia, (7) right ventricular hypertrophy, (8) right ventricular failure. It is worthy of note that this case fulfills all eight of these criteria.

The largest series of cases of this type to date is six reported by Estes et al.[3] They postulate that hypoventilation leads to hypercapnia and hypoxia and that these contribute to the development of polycythemia, pulmonary hypertension, right-sided cardiac enlargement, and ultimately to heart failure. This group, in common with all the others reporting a similar entity, feels that the symptoms and findings are related to obesity. These findings are generally reversible and the key to reversal is weight reduction.

* Presented at the Seventy-eighth Annual Meeting of the Louisiana State Medical Society, Shreveport, May 7, 1958.

CASE REPORT

The patient, a 53 year old white male, was first seen at the North Louisiana Clinic in 1950, at which time he gave a history of swelling of the abdomen of several months duration. He had a history of chronic alcoholism intermittently since 1923, and drank particularly heavy during a period from

1923 to 1931. Bromsulfalein excretion was markedly impaired and he had fasting blood sugars which were always slightly elevated. His weight, in 1950, was 260 pounds, representing an increase of 100 pounds since 1932. He was considered at that time to have hepatic insufficiency and was treated with diet and insulin and was digitalized with apparent benefit.

He responded well to the conservative management and was not seen again until January 8, 1957, when he gave a history of having developed marked shortness of breath in April 1956. He saw a local physician who advised hospitalization but he decided to go to a Clinic in another city for evaluation. He was detained at the other group for five weeks and was told that he had asthma and placed on some form of inhalation therapy which he stated helped his shortness of breath. He has had a history of chronic bronchial irritation with frequent attacks of bronchitis and occasional coughing and wheezing all of his life and stated he had never been able to breathe really well. However, in January 1957, he noted a marked sense of fullness and soreness in the right side along with steadily increasing dyspnea.

Rales were heard at both bases and scattered wheezes were heard throughout the chest. He had 3 plus edema of the lower extremities. His blood pressure was 210/-136 and urinalysis at that time revealed 1 plus albumin with coarsely and finely granular casts. No sugar was found in the urine. Fasting blood sugar was 123 mgm. per cent, NPN 27 mgm. per cent. Total proteins were 5 grams with 3.2 of albumin and he had a BSP retention of only 2.5 per cent in 45 minutes. His prothrombin time at that time was 85 per cent and he had a negative cephalin cholesterol flocculation. Weight was 282 lbs.

He was taking 30 mgm. of Benzadrine each morning in order to stay awake and he exhibited marked evidence of cyanosis with extreme changes appearing in the nail beds and around the face. Orthopnea was so profound at this time that he was able to sleep only sitting in a chair. This was improved somewhat by stopping his Benzedrine but orthopnea gradually became worse. Repeated blood counts showed him to have as high as 6.2 million RBC's, hemoglobin at that time running well above 100 per cent.

The possibility of Cushing's syndrome was considered but was not substantiated. He was referred to the Lung Station at Confederate Memorial Medical Center where studies revealed findings shown in Table 1.

On fluoroscopy the diaphragms were elevated by the abdominal obesity and there was evidence of slowed diaphragmatic motion. The impression was that of mild obstructive ventilatory disease.

The marked desaturation of the arterial blood at rest did not seem consistent with the degree of ventilatory impairment with a normal ventilation equivalent.

TABLE 1.

	Observed	Predicted
Maximal breathing capacity	38-1/m	Over 124/m
Vital capacity	1800 cc	3790 cc
Breathing reserve	77%	95%
Air velocity index	.65	1.00
Aretrial O_2 saturation:		
Resting breathing room air	60.5%	96%
Resting breathing pure O_2 for 10 min.	95.5%	100%

The failure of the patient to saturate the blood after 100 per cent 0_2 seemed to indicate a venous-arterial shunt.

He was placed on a 1200 calorie reducing diet with supplementary vitamins, was digitalized cautiously and was treated with a hypotensive agent. The diet was salt free. He did fairly well, although he continued to have intermittent episodes of edema. On July 1, 1957, he was markedly edematous. At that time he had an albumin fraction of only 2.56 with a globulin of 2.21, and in view of the steadily increasing edema along with a cardiac rate of 88 per min. he was given an ampule of salt free serum albumin. Following this he felt markedly improved for several days and urinary output was satisfactory, but then he began to fill up again and was extremely uncomfortable. His weight was back up to 298 lbs. He

exhibited 4 plus edema of the lower extremities, marked edema of the abdominal wall and dyspnea on minimal exertion with orthopnea to the point where it was impossible for him to rest in any position other than an erect one. At that time, he was referred to Dr. Edgar Hull in New Orleans and was seen during his stay in New Orleans by Drs. Hull and Garcia. At this point I will ask Dr. Garcia to fill in the part of the picture dealing with his hospitalization in New Orleans.

Dr. John E. Garcia (New Orleans): The patient entered Mercy Hospital on August 2, 1957, with a history as given by Dr. Herold. On physical examination he was seen to be very obese, weighing 310 lbs. on admission. Lips and face were deeply cyanotic. The lung fields were clear, the heart regular, with a blood pressure of 160/90. Auscultation of the heart revealed the presence of a systolic and diastolic murmur best heard in the 2nd and 3rd anterior interspaces along the left sternal border. Flouroscopy and x-ray of the chest revealed marked cardiac enlargement due to right ventricular hypertrophy. The electrocardiogram was within normal limits, showing only digitalis effect and an occasional ventricular premature beat. The abdomen was very prominent and ascites was considered likely. There was marked edema of the lower extremities and subcutaneous tissues of the abdomen extending to the costal margin.

Laboratory findings on admission: Red blood cells 5.6 million with 16 gms. of Hgb., hematocrit 55, normal white blood cells and differential, blood glucose 114, blood urea nitrogen 45, creatinine 1.0. The urine contained 4 plus albumin and hyaline and granular casts. The BUN a few days after admission dropped to 33, the CO_2 combining power was 63 vol. % (28 meq.), chlorides 100 meq. (580 mgm. %), K 5.5 meq. and NA 143 meq.

It was noted that the patient became very drowsy and would fall asleep readily (while being examined, or even during conversation.) He was easily awakened and while awake was perfectly lucid, not at any time being really stuporous or mentally confused. Jerky, muscular twitchings were also observed from time to time.

Treatment in the beginning consisted of digitalis, ammonium chloride, mercurial diuretics and a low salt 1,000 calorie diet. Abdominal paracentesis was attempted three times, a small amount of fluid being obtained on the third attempt by using a very long needle. Response to this therapy was slow and ineffective and it was felt that more radical measures would be necessary. Upon the suggestion of Drs. Edgar Hull and Louis Monte 275 mgm. of cortisone daily was begun on August 12, 1957, and was continued for the following eighteen days, his diet at the same time being reduced to 800 calories a day. It was at once apparent that the cortisone was exerting a beneficial effect, producing a marked diuresis and accordingly a continuous and dramatic loss of weight. At the end of eighteen days the dosage of cortisone was reduced to 200 mgm. a day and then gradually to 100 mgm. a day. During this time his entire clinical picture was one of improvement, with complete loss of edema, change in appearance and a loss of 71 lbs. in weight. Cyanosis improved and the muscular twitching disappeared altogether. No adverse effects were produced by the cortisone. He was discharged from the hospital on September 6, 1957, with a diagnosis of cor pulmonale, obesity, and secondary polycythemia, which make up the clinical condition known as the Pickwickian Syndrome.

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DISCUSSION

Dr. John E. Garcia (New Orleans): This syndrome is called the Pickwickian syndrome because of the resemblance of the patient to the "fat and red-faced boy in a state of somnolency" as described by Charles Dickens. This same clinical entity has been variously described as "Extreme obesity associated with alveolar hypoventilation"; "A cardiopulmonary syndrome associated with extreme obesity"; "Obesity, polycythemia and alveolar hypoventilation"; "Progressive respiratory

distress"; "Cardiopulmonary syndrome caused by obesity"; and "Obesity heart disease". It is obvious that obesity is the common factor and that weight reduction is important, because many aspects of this syndrome can be reversed by a sufficient loss of body weight.

In this particular patient cortisone was used as a diuretic, as has been reported in cases of refractory edema due to heart failure. We believe that this is the first time the use of cortisone has been reported in this particular syndrome. As further evidence of its efficacy we have used cortisone successfully in two additional patients with typical features of the "Pickwickian Syndrome." Both of these patients, incidentally, are females. The exact mode of action of cortisone is obscure.

All of the credit for success in treatment cannot of course be attributed to cortisone. The usual cardiac failure regimen was employed together with a restricted diet and oral diuretics. Diuril was not yet on the market at the time but is now being used on the other two patients.

The patient under discussion now weighs 235 lbs., feels well, has no edema or shortness of breath, and his RBC is down to 4.5 million with 13.5 gms. of Hgb.

THE PRESENT STATUS OF MUSCLE RELAXANTS *

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Search continues for the ideal muscle relaxant since Sir Walter Raleigh [1] first mentioned discovery of Guiana in 1595. The botanical sources of the various curares have never been satisfactorily identified nor have their active ingredients ever been adequately analyzed. Some semblance of order in this chaotic complex was obtained in 1897 by Boehm,[2] who distinguished the curares, depending on the container in which they were available, as tube, pot, and calabash. Real progress in the clinical application of curare was made possible by the cooperation of several investigators. McIntyre and associates [3] prepared first a purified curare extract from crude curare brought to the United States by Gill.[4] Curare was first used for anesthesia by Griffith and Johnson,[5] who at the suggestion of L. H. Wright administered it in the form of Intocostrin® to anesthetized patients.

The synthetic compound, succinyldicholine, which approaches more closely the ideal muscle relaxant, has been lying idly on the shelves of chemists since it was first prepared by Hunt and Taveau in 1906.[6] Forty-three years later, Bovet and associates [7] discovered the neuromuscular activity of this compound. In recent years numerous agents have been synthetized. Some of these, for example, suxethonium (Brevedil "E") and laudexium (Laudolissen®), have been used clinically.

The main ingredient of the various curares made by the Indians of South America was either the bark of one of the many varieties of the genus strychnos, most frequently *Strychnos toxifera*, or the vines of *chondrodendron tomentosum*.

CHEMISTRY

Boehm [2] was the first to approach systematically the problem of isolation of curare alkaloids. He isolated quaternary ammonium bases called curarines from the three available varieties of crude curare. According to their source of origin, he named the products obtained from calabash curare, curarines, those from pot curare, protocurines, and those from tube curare, tubocurarines. He also isolated from pot and tube curare tertiary bases with weak neuromuscular activity which he termed curines.

Of the numerous alkaloids isolated from calabash curare, the most interesting is toxiferine-I, which although perhaps the most potent of all neuromuscular blocking agents, contains only one quaternary ammonium group.

Another naturally occurring alkaloid

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with neuromuscular blocking activity is B-erythroidine. In contrast to the curare alkaloids this compound is a tertiary amino derivative and contains no quaternary ammonium groups. Quaternary derivatives of naturally occurring cinchona, pyridine, isoquinoline, and tropine alkaloids are also capable of interrupting neuromuscular transmission.

Crum-Brown and Fraser [8] were the first to attempt to produce synthetic muscle relaxants by synthesis of quaternary methylstrychnine and methylbrucine derivatives. More recent efforts in this direction have centered around the importance of optimally placed quaternary ammonium groups. The muscle relaxants used most frequently in clinical practice all contain two or more quaternary ammonium groups. These groups, whether separated by a straight chain, as in decamethonium or succinylcholine, or by aromatic radicals, as in benzoquinonium, gallamine or laudexium, are about 15 Angstrom units from one another.

In general, converting a tertiary nitrogen compound into a quaternary one will increase neuromuscular activity. Substitution of the methyl radicals in the quaternary ammonium groups with ethyl radicals or with nitrobenzyl radicals changes certain depolarizing muscle relaxants to nondepolarizing agents.

PHYSIOLOGY

After the motor nerve fiber penetrates the sarcolemma of the muscle fiber, it loses its myelin sheath and the neurilemma blends with the sarcolemma. The axoplasm spreads out in close contact with the sarcolemma and it is assumed that the side facing the muscle fiber is covered by the terminal membrane. The terminal membrane is separated by a microscopic gap, the subneural space, from the sole plate of Kuhne. Morphologically, the sole plate is modified sarcoplasm of the muscle fiber. The side of the sole plate facing the terminal membrane is covered by the postsynaptic or postjunctional membrane. The subneural space is therefore limited on one side by the terminal membrane of the motor nerve ending and on the other by the postjunctional membrane.

The essentials of neuromuscular transmission, which according to Nachmansohn as cited by Wilson,[9] are basically not different from axonal conduction, can be summarized as follows:

The postjunctional membrane contains, acetylcholine, four different types of proteins associated with acetylcholine metabolism and various ions. The resting postjunctional membrane is polarized. The interior of the postjunctional membrane is rich in potassium ion and poor in sodium ion, and is electronegative with reference to the outer surface, which is rich in sodium and poor in potassium.

The electrostatic difference between the outer surface and the interior of the resting end-plate is about -90 millivolts. This is called the resting membrane potential.

When the nerve impulse reaches the myoneural junction, acetylcholine is liberated from its protein bondage. The liberated acetylcholine is adsorbed immediately to the second protein, which is also known as the cholinergic receptor of the end-plate. Owing to the adsorption of acetylcholine to it, the cholinergic receptor changes its configuration and thereby increases the permeability of the postjunctional membrane for sodium and potassium ions. Potassium diffuses out of, and sodium diffuses into, the end-plate and the potential difference between the interior and exterior decreases. This rapid change in the negativity was termed "end-plate potential" by Eccles and coworkers.[10] This potential change in about 0.3 milliseconds decreases the potential to -45 millivolts. At this stage, there is no spread to muscle fiber. When this stage is surpassed, the end-plate potential becomes action potential.

The end-plate remains depolarized for only 2 to 3 milliseconds because shortly after its adsorption to the cholinergic receptors, acetylcholine is attracted to the third protein, the acetylcholine esterase, present in the postjunctional membrane, and is rapidly hydrolyzed to acetic acid and choline. Simultaneously the postjunctional membrane becomes repolarized and the membrane potential of the end-plate returns to its resting level of -90 millivolts.

Subsequently the hydrolysis products of acetylcholine, choline and acetic acid, are resynthesized by the fourth protein, called choline acetylase, to acetylcholine which is adsorbed to the storage protein, thereby completing the cycle.

Anything that will interfere with either the depolarization or the repolarization of the end-plate will inhibit neuromuscular transmission.

Nondepolarizing and depolarizing drugs compete with acetylcholine for the cholinergic receptors of the end-plate and are capable of preventing the access of acetylcholine to these receptors partly because of their greater stability and partly because of their greater affinity for these receptors. After being adsorbed to the receptor proteins, the members of the two groups behave differently.

The nondepolarizing agents play a passive role, and their activity is limited to the prevention of access of acetylcholine to the receptors.

The depolarizing agents, like acetylcholine, cause depolarization of the end-plate but depolarization persists, spreads to the fiber adjacent to the end-plate and renders the muscle fiber insensitive to subsequent indirect stimuli.

The neuromuscular block caused by depolarizing agents produces flaccid paralysis which is preceded by transient stimulation in mammalian muscle.

The different depolarizing muscle relaxants have an additive effect and their activity is antagonized by nondepolarizing muscle relaxants.

Ether and cyclopropane do not potentiate the depolarizing agents. Procaine, administered before, antagonizes, but administered after, potentiates the block caused by depolarizing agents. Acetylcholine and anticholinesterases increase blocking activity. Edrophonium has either no effect or potentiates depolarization block.

Potassium, anoxia or prior tetanization does not antagonize the block. Anodal current antagonizes and cathodal current potentiates depolarizing agents.

PHARMACOLOGY

McIntyre [11] observed that curare in subparalytic doses caused an increase, then a decrease, in the electroencephalographic activity of dogs. Ellis and associates [12] found that decamethonium and succinylcholine had an effect on the respiratory center similar to that of d-tubocurarine. The central effect of succinylcholine seemed less pronounced than that of d-tubocurarine and decamethonium.

Muscle relaxants can influence respiration by paralyzing respiratory muscles, by depressing the activity of the respiratory center, by influencing the chemoreceptors of the carotid body, and by bronchoconstriction due to histamine release.

The autonomic effects of muscle relaxants are due to substrate competition with acetylcholine for the cholinergic receptors of the autonomic ganglia. Of the clinically used muscle relaxants, d-tubocurarine, possesses the greatest autonomic activity.

Of the clinically used muscle relaxants, d-tubocurarine exhibits the most pronounced effects on the cardiovascular system. The cardiac sympathetics, however, were found to be ten times as resistant to d-tubocurarine as the myoneural junction. d-Tubocurarine and gallamine both exhibited a protective action against epinephrine induced cardiac arrythmias in cats anesthetized with cyclopropane.

Decreased peristalsis and intestinal distention were reported after paralyzing doses of d-tubocurarine. Gallamine, benzoquinonium, decamethonium and succinylcholine have little effect on tone and motility of the intestinal tract.

Release of histamine is more prone to occur with d-tubocurarine than with any of the other clinically used muscle relaxants.

Cholinesterase is inhibited by d-tubocurarine, gallamine, benzoquinonium, decamethonium and succinylcholine.

Formation of urine was reported to be diminished by d-tubocurarine in dogs.[13] The clinically used muscle relaxants are excreted partly or wholly unchanged in the urine. Because of hydrolysis only 5 to 15 per cent of succinylcholine administered

intravenously was found to be excreted unchanged in the urine of cats and mice.

Uterine contractions were not affected by paralyzing doses of d-tubocurarine in dogs.[14]

It has been recently reported that the intravenous administration of succinylcholine causes sustained elevation of the intraocular pressure in conscious human volunteers [15] and anesthetized man.[16] Mammalian eye muscles may react to succinylcholine with contracture, that is, nonpropagated sustained contraction localized to restricted regions of the fibers, instead of contraction.

The action of muscle relaxants is considerably prolonged when they are administered by other than the intravenous route.

Without artificial respiration the primary cause of death is respiratory arrest.

USES

The most common uses of muscle relaxants are for endotracheal intubation, endoscopy, supplementation of general anesthetics, treatment of laryngospasm, abdominopelvic examination and reduction of dislocations and fractures. Muscle relaxants are also used in convulsive shock therapy, treatment of certain diseases such as Parkinsonism and tetanus, and diagnosis of such diseases as myasthenia gravis.

COMPLICATIONS

Improper selection or administration of excessive doses of the muscle relaxant may lead to serious complications that occasionally may endanger the patient's life. In general, these complications are due to: (a) excessive and prolonged action at the neuromuscular junction; (b) their effect on ganglionic autonomic transmission, or (c) their ability to liberate histamine from the tissues. The complications may occur both during anesthetization and after the effects of the anesthetic have worn off and can affect the respiratory and circulatory systems, the gastrointestinal tract or the voluntary muscles.

Hypotension is caused by ganglionic blocking action, release of histamine, or a combination of these two factors. The incidence is low and its intensity is seldom great.

The elevation of blood pressure associated with administration of succinylcholine can be effectively counteracted with hexamethonium. Administration of gallamine almost invariably causes acceleration of the heart rate. In contrast, use of benzoquinonium occasionally causes bradycardia.

The effect of muscle relaxants on the myocardium is negligible. Depending on the relaxant used, 200 to 1500 times the paralyzing dose can be administered intravenously before the development of cardiac arrest. It is, therefore, safe to assume that inadequate oxygenation or myocardial depression caused by excessive concentrations of the general anesthetic agent should be held responsible for cardiac arrest.

ANTAGONISTS

With the correct selection and method of administration of muscle relaxants, use of antagonists should seldom be necessary. Doughty and Wylie [17] and Hunter [18] suggested the use of atropine sulphate with neostigmine. Edrophonium is less effective in deeply curarized patients than in those in whom the effect of the muscle relaxant has started to wear off. Larger doses are required when ether, cyclopropane, or nitrous oxide is used.

The interval between the intravenous injection and the development of effect is shorter for edrophonium than for neostigmine. The duration of action of edrophonium is also shorter than that of neostigmine.

Theoretically, there are at least three possibilities for cessation of neuromuscular block caused by nondepolarizing agents.

1. The concentration of the agent at the end-plate decreases by redistribution due to excretion, decomposition or uptake by other tissues below a level at which the acetylcholine present in physiologic concentrations at the end-plate can generate end-plate potential of sufficient intensity to initiate muscular contraction. This is the natural course of events.

2. The concentration of acetylcholine at the end-plate can be raised by the use of cholinesterase inhibitors (e.g., neostigmine). The increased number of acetylcholine molecules will displace the molecules of the blocking agents from the receptors,

and the released molecules are carried away by the circulation.

3. The molecules of the nondepolarizing blocking agents may be displaced by molecules of depolarizing substances (e.g., edrophonium) which have a greater affinity for the end-plate receptors than the nondepolarizing blocking agents. Here again the released relaxant molecules are removed by the circulation.

. The side effects and possible complications of both neostigmine and edrophonium are due primarily to their muscarinic properties, caused by the action of an excess of acetylcholine on the postganglionic component of the parasympathetic system.

The greatest danger reported after the use of neostigmine is pronounced slowing of the heart rate, which in extreme cases may cause cardiac arrest. Bradycardia is less pronounced with edrophonium, especially if general anesthesia is maintained by ether. Excessive salivation and bronchial secretion, bronchiolar spasm and intestinal hypermotility are pronounced after neostigmine. Intestinal hypermotility is especially dangerous after gastrointestinal operations. Another complication is recurarization in a seemingly recovered patient who is left unobserved.

CONCLUSIONS

Of the presently available muscle relaxants, with the exception of few situations, succinylcholine is the drug of choice. However, in at least two respects succinylcholine falls short of the pharmacologically ideal muscle relaxant. One of these is that the block produced by succinylcholine is depolarizing and, therefore, the neuromuscular junction is not in the physiologic resting state while under its influence. The second disadvantage is that its primary breakdown product, succinylmonocholine, also has some neuromuscular activity and, therefore in certain circumstances the return of normal respiratory activity may be delayed after its use.

The type of block obtained with succinylcholine depends upon the properties of the receptor protein of the species involved. In some species, in which the configuration of the receptor protein can be changed easily, relatively small doses produce depolarization block. In those in which the receptor proteins are more resistant to configuration changes, small doses produce no neuromuscular block and large doses cause nondepolarizing block.

A good example of the decreased sensitivity of the end-plate to depolarization is seen in patients with myasthenia gravis in whom depolarization relaxants produce a nondepolarizing block.

The ideal muscle relaxant should be nondepolarizing, short-acting and controllable as succinylcholine; its fate in the organism should be little affected by pathologic changes, its breakdown products should have no neuromuscular blocking effect and it should be easily reversible by a harmless antagonist.

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THE USE OF OMNADIN * IN REPEATED AND CHRONIC INFECTIONS ASSOCIATED WITH A LOW SERUM GAMMA GLOBULIN

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A preliminary report on the use of Omnadin* a brand of prolipin lipoprotein compound in cases with repeated and chronic infections associated with a low blood serum gamma globulin is being presented. This study was prompted by the fact that many patients who have been having recurrent upper respiratory infections associated with low grade fever of undetermined origin often show by serum paper electrophoresis a decrease from the normal range of the gamma fraction of the blood. The exact normal value for this particular globulin fraction has not been fully established in the literature, but, from the statistics of this laboratory, the normal range has been set between 0.80 gms.% and 1.5 gms.%

Upon investigation of this type of patient with the gamma globulin level below 0.80 gms.%, it was suggested that the decrease could be a result of the concurrent disease or that some deficiency in the immune mechanism was present. Since it is a known fact that gamma globulin plays an important role in antibody formation, an attempt was made to find a method to raise the gamma globulin level. Inasmuch as gamma globulin per se is a costly medication and produces only a short lived passive immunity, a less expensive substance, Omnadin, was used with the hope of producing, or of stimulating, an active immunity.

With this thought in mind several selected cases were started on Omnadin with the following dose schedule:

1 cc. daily for five days, subcutaneously

1 cc. three times weekly for one week, subcutaneously

1 cc. twice weekly for four weeks, subcutaneously

1 cc. once a week for four weeks, subcutaneously

1 cc. monthly for several months, subcutaneously

*A brand of prolipin lipoprotein compound, supplied by Winthrop Laboratories for this investigation.

CASE REPORTS

A review of these cases is as follows:

Case No. 1-A.P., white female, followed from birth to 16 months. She had numerous bouts of tonsilitis, nasopharyngitis, pyelitis, and the infectious childhood diseases, measles and chickenpox. These required numerous medications, including penicillin, sulfa, and tetracycline drugs. For a ten month period following administration of Omnadin, the child had only one bout of otitis media and received one round of penicillin therapy for this infection. There was a 13 pound weight gain during this period.

Case No. 2-B.N., white male, was followed from birth to 4½ years of age. He was seen in the office on numerous occasions with allergic dermatitis, nasopharyngitis, tonsillitis, along with otitis media. He was hospitalized six times during this period for infections, and received antibiotics during each bout of illness. During the eighteen months after the administration of Omnadin he was seen one time with tonsillitis and one time with allergic dermatitis. Eight pounds of weight were gained during this period.

Case No. 3-S.J., white female, was followed from birth to 4½ years of age. She had measles, chickenpox, and numerous bouts of upper respiratory infection. Each infection required antibiotic therapy. She has been under observation for nine months after Omnadin therapy and has had no infections during that period of time.

Case No. 4-R.D., white male, with a follow-up from 8 months to 1½ years of age. The past history revealed bronchopneumonia on 4 occasions, bouts of tonsillitis, nasopharyngitis with otis media. He received all types of antibiotic therapy during this period of time. He has been seen during a ten month period after administration of Omnadin, has had no infections and has gained 10½ lbs.

Case No. 5-R.B., white male had been under observation from the age of 4 to 6 years. Prior to the administration of Omnadin, he was treated for bronchopneumonia twice, otitis media on 4 occasions, nasopharyngitis on 1 occasion, tonsillitis on 3 occasions, and received antibiotic therapy for each infection. During an eight month period follow-up after administration of Omnadin the patient had two bouts of tonsillitis with otitis

TABLE 1

Case No.	Total Protein	Albumin	Globulin	A/G Ratio	Alpha I	Alpha II	Beta	Gamma	Date
1	6.15	3.98	2.17	1.8	0.28	0.76	0.52	0.62	3-31-56
	5.90	3.37	2.56	1.3	0.28	0.57	0.81	0.87	6-18-56
2	6.72	3.43	3.29	1.1	0.31	1.14	1.32	0.52	7- 9-56
	6.82	3.23	3.59	0.9	0.41	1.26	0.94	0.98	1- 5-57
3	6.29	3.91	2.38	1.6	0.46	0.55	0.78	0.60	7-12-57
	6.65	3.81	2.84	1.34	0.29	0.44	1.10	1.01	10- 2-57
4	6.12	2.74	3.38	0.8	0.21	1.57	1.0	0.61	8-30-56
	6.73	3.50	3.23	1.1	0.30	0.77	1.07	1.09	10- 3-56
5	6.23	2.93	3.30	0.9	0.66	1.14	0.88	0.62	10- 6-56
	7.32	3.40	3.92	0.9	0.36	1.22	1.25	1.02	1-15-57
6	6.25	3.48	2.77	1.2	0.37	1.01	0.89	0.50	12-19-56
	6.29	3.46	2.83	1.2	0.43	0.61	1.07	0.72	3-12-57
7	7.00	4.05	2.95	1.4	0.39	1.17	0.68	0.71	10- 3-56
	7.41	4.28	3.13	1.37	0.40	0.70	1.06	0.90	1-23-57

media and appendicitis. He did receive penicillin on one occasion and achromycin on another occasion, 5 lbs. weight gained.

Case No. 6-J.S., white female was followed from birth to 6 months. Prior to Omnadin, she had bronchopneumonia on 2 occasions, numerous bouts of tonsillitis, nasopharyngitis, upper respiratory infections and received antibiotic therapy on each occasion. For a nine month period following the administration of Omnadin, she was treated one time for a bout of tonsillitis. She gained 7 lbs. weight.

Case No. 7-B.G., white female was followed from birth to 1 year of age. She had numerous bouts of upper respiratory infection with otitis media and bronchopneumonia, and received antibiotic therapy with each bout of infection. For a nine month period following the administration of Omnadin she has had one upper respiratory infection and one bout with tonsillitis. Received antibiotic therapy with tonsillitis. Weight gained approximately 6 lbs.

Above is a summary of 7 patients who were put on the prescribed dose schedule. These 7 cases are still receiving a monthly injection of 1 cc. of Omnadin upon insistence of the parents who have been impressed with the results.

It is apparent from the above case studies as noted in Table 1 that there is a rise in the gamma globulin level concomitant with the administration of Omnadin. This fact may be coincidental but may represent a rise in the antibody containing fraction of the gamma globulin (stimulated by the drug). Marked subjective as well as objective improvement was noted in all 7 cases reported.

Omnadin has also been administered to approximately 35 other patients, some of whom have had a relatively normal gamma globulin level and in others electrophoretic study was not made. The rather broad indications for the use of Omnadin were chronic repeated infections, particularly of the throat, lungs, and skin, which had not responded to the usual forms of conventional therapy. There are no figures available as to the exact results obtained, but a conservative estimate would show that 80 to 90 per cent of these patients had moderate improvement after receiving the lipoprotein.

Approximately 1500 injections of Omnadin have been given to date without any sign of local or systemic undesirable side effect.

CONCLUSION

The case studies summarized above suggest that there is a possible relationship between hypogammaglobulinemia states and repeated infections associated with a faulty mechanism for providing active immunity. The use of Omnadin for this condition appears to raise the gamma globulin fraction and possibly in doing so enhances the antigen-antibody response.

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A.M.A. ADOPTS NATIONAL INTERPROFESSIONAL CODE FOR PHYSICIANS AND ATTORNEYS

The House of Delegates of the American Medical Association, at its Annual Meeting in San Francisco, in 1958, adopted a new national interprofessional code for physicians and attorneys. This code is the work of a joint national medical-legal liaison committee, the appointment of which was requested in 1957 by the president of the American Bar Association. It represents a year's effort in which conferences have been held and the code has been formulated after a thorough study of the different codes which have been adopted by bar associations and medical societies in various areas at the state and local levels. This code was intentionally prepared in general terms to permit its adaptation in the light of local conditions and circumstances.

The provisions of the code are not to be regarded as laws but suggested rules of conduct for the members of the two professions. It is still subject to the principles of medical and legal ethics and the rules of law prescribed for the individual conduct. The code supplies guides for physicians and attorneys in their interrelated practice in the area covered by its provisions.

Its purpose was stated as an effort to promote the public welfare, improve the practical working relationships of the two professions, and facilitate the administration of justice.

The relative importance of medical testimony has increased in litigation to the extent that now seven out of every ten cases involve medical testimony, and therefore, the presence of a physician. The duty of the physician is the same as that of any other person to respond to judicial process. It is also his duty to supply the medical facts relevant to a legal contest when they are required by the processes of justice. The code states that the attorney and physician should treat one another with dignity and respect in the courtroom; that the physician who testifies as to medical facts should never be an advocate, and should realize that his testimony is intended to enlighten rather than to impress or prejudice the court or the jury.

The various sections of the code cover such topics as medical reports, pretrial conferences, the issuing of subpoenas, and the prior notification to the physician by the attorney of the subpoena, arrangements for court appearances, and the proper conduct of the physician and of the attorney when the physician is a witness. Sections also give consideration to fees for

the services of the physician relative to the litigation, and payment of medical fees. The code advises that it be implemented at state and local levels for the purpose of improving the interprofessional relationships between the legal and medical professions. Provision is made for the handling of complaints on the part of either attorney or physician through appropriate channels, and deplores public airing of such complaints. The general trend of the code is to make the physician feel that it is his duty to cooperate in these legal processes, and he should recognize that delays in providing medical information may prejudice many aspects of the case and defeat the ends of justice.

The liaison committee of the two national associations also gave consideration to the encouragement of state and local medical-legal meetings, to medical professional liability problems, medical-legal forms, and the possibility of establishing medical-legal courses in law schools and medical schools.

The reluctance of the physician to go into court is understandable. He is concerned in his day to day work with the shifting effects of nature's forces. The operation of these processes of nature is only partly known to him. He is never in possession of all the facts of a case, and medicine is not an exact science. The knowledge that he may have is not readily reduced to writing, and even then only with considerable expense and difficulty. It is doubtful if there ever was a situation in which the physician as a witness had written records sufficient to answer all the questions of opposing attorneys.

The other principal reason for a physician wishing to avoid serving as a witness is the attitude frequently exhibited by attorneys in a manner expressed or implied that the physician should represent all knowledge or be master of the whole field of medicine. While this is obviously fantastic, the physician's limitations may be displayed in the courtroom in a light which is humiliating.

The code of ethics expressly makes the effort to protect the physician in these two fields, and states that the rules of evidence when adequately utilized by the court offer ample opportunity to test the qualifications, competence, and credibility of a medical witness.

The code is an excellent guide to improving interprofessional relationships between physicians and attorneys. All should read it and it will be helpful in proportion to the extent that it is implemented by local societies.

—o—

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The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

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J. P. Sanders, M.D., Chairman, Shreveport; J. T. Cappel, M.D., Alexandria; B. F. Mancuso, M.D., New Orleans; Guy R. Jones, M.D., Lockport; M. C. Wiginton, M.D., Hammond; Barclay Funk, M.D., Lake Charles.

STATE HOSPITAL POLICIES AND MEDICAL INDIGENCY

P. H. Jones, M.D., Chairman, New Orleans; George W. Wright, M.D., Monroe; T. A. Kimbrough, M.D., Lafayette; Charles B. Odom, M.D., New Orleans; B. E. Trichel, M.D., Shreveport; E. L. Zander, M.D., New Orleans.

WOMAN'S AUXILIARY (ADVISORY COMMITTEE)

J. Y. Garber, M.D., Chairman, Lake Charles; J. T. Willis, M.D., Alexandria; E. L. Leckert, M.D.; C. Grenes Cole, M.D.; both of New Orleans.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Ascension	Third Tuesday of every month	
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

AIR FORCE GENERAL PRACTICE RESIDENCY PROGRAM

Approved residencies in general practice are available at the USAF Hospital, Maxwell Air Force Base, Alabama, and at certain civilian hospitals. This program provides one year of training in medicine, including pediatrics and psychiatry; and one year in surgery including traumatic surgery, fractures, obstetrics and gynecology.

It is anticipated that programs will be approved by July 1959, in additional hospitals at Andrews Air Force Base, Washington, D. C.; Keesler Air Force Base, Mississippi; and Wright-Patterson Air Force Base, Ohio. After completing residency training in general practice, medical officers may attend the primary course in aviation medicine,

if they so desire, and are physically qualified.

Physicians interested in an Air Force career in general practice may write directly to The Surgeon General, Headquarters, USAF, Washington 25, D. C.

BIBLIOGRAPHY AVAILABLE

The National Library of Medicine has compiled a lengthy bibliography on staphylococcal infection, which, it is hoped, may be of value to both private and public health physicians who are engaged in combatting the increased incidence of antibiotic resistant staphylococcal infection in the home, community and hospital. The bibliography will be sent at no cost on request to the National Library of Medicine, 7th St. and Independence Ave., S. W. Washington 25, D. C.

TENNESSEE VALLEY MEDICAL ASSEMBLY

The Read House, Chattanooga, Tennessee will be headquarters for the Tennessee Valley Medical Assembly to be held September 29-30. Participating in the program will be Drs. William R. Arrowsmith and Merrill O. Hines, of New Orleans. Dr. Arrowsmith will speak on "Present Management of the Lymphomas" and the subject to be presented by Dr. Hines is "Lesions of the Colon with a Negative Colon X-ray."

GASTROENTEROLOGICAL CONVENTION

The twenty-third annual convention of the American College of Gastroenterology will be held at the Jung Hotel in New Orleans, October 20-22. In addition to the many individual papers to be presented, there will be panel discussion on gastric carcinoma, steroids in gastroenterology and functional disturbances of the gastrointestinal tract. There will also be scientific as well as commercial exhibits and the sessions will be open to all physicians without charge.

On October 23-25, immediately following the convention, Dr. Owen H. Wangensteen of Minneapolis, Minnesota, and Dr. I. Snapper of Brooklyn, New York, will be the moderators of the annual course in postgraduate gastroenterology. The sessions will be held at the Jung Hotel and in the auditorium of the Louisiana State University School of Medicine. Attendance at the course will be limited to those who have registered in advance.

Copies of the program and further information concerning the postgraduate course may be obtained by writing to the American College of Gastroenterology, 33 West 60th Street, New York 23, N. Y.

SYMPOSIUM ON CARCINOMA OF THE COLON AND RECTUM

The American Cancer Society will hold a scientific session at the Biltmore Hotel, New York City, on October 20-21, at which time there will be presentation of papers and panel discussions on the subject of cancer of the colon and rectum. Inquiries concerning this program should be addressed to the Director of Professional Education, American Cancer Society, Inc., 521 West 57th Street, New York 19, N. Y.

AMERICAN PUBLIC HEALTH ASSOCIATION

The eighty-sixth annual meeting of the American Public Health Association will be held in St. Louis, October 27-31. This meeting will feature reports on progress being made by voluntary and governmental agencies toward solving the major health problems facing mankind. These problems include ionizing radiation and atmospheric pollution, dental health, engineering and sanitation, epidemiology, food and nutrition, health officers, role of the public health laboratory, maternal and child health, mental health, occupational health, public health education, public health nursing, school health, and statistics marking the fiftieth anniversary of the Section on Statistics.

AMERICAN RHINOLOGIC SOCIETY MEETING

The American Rhinologic Society will hold its fourth annual meeting at the Palmer House, Chicago, October 17-18. Among the topics to be discussed will be pulmonary and nasal physiology, laboratory and clinical aspects of bone transplants, hump removal, roof repair, and nasal process corrections. The preliminary program includes a paper on "Maxillary and Premaxillary Approach to Septal Surgery," by Dr. Ralph H. Riggs, of Shreveport.

The Society will display many reprints, papers, slides, charts and other teaching and study material available to the profession upon request. Two new exhibits suitable for showing at state society and other professional meetings will also be presented.

There is no registration fee and all members of the medical profession are invited to attend as guests. For further information write to Dr. Robert M. Hansen, Secretary of the Society, 1735 North Wheeler Avenue, Portland 17, Oregon.

APPOINTMENT OF DR. FOX

Dr. John P. Fox, professor of epidemiology, Tulane School of Medicine, has been appointed Director of the Division of Graduate Public Health at Tulane. In this capacity he will become the administrator of Tulane's graduate training program in public health and tropical medicine. Dr. Fox has also been designated to become the first occupant of The William Hamilton Watkins chair in epidemiology established by Mr. and Mrs. Charles E. Inbusch of Milwaukee, Wisconsin. Tulane's degree-granting program in public health and tropical medicine, established in 1947, is one of twelve in North America and is participated in by students from all parts of the United States as well as the world.

NEWS FROM OCHSNER FOUNDATION HOSPITAL, NEW ORLEANS

Dr. Patrick H. Hanley, New Orleans, was elected Vice-president of the American Proctologic Society at its meeting, July 1-3, in Los Angeles.

Dr. Paul T. DeCamp, New Orleans, was elected Secretary of the North American Chapter, International Cardiovascular Society at its meeting, June 21, in San Francisco.

AMERICAN ASSOCIATION OF MEDICAL ASSISTANTS

All medical assistants are cordially invited to attend the Second Annual Meeting of the American Association of Medical Assistants, October 31, November 1 and 2, 1958, Palmer House, Chicago, Illinois. We urge each physician to call this meeting to the attention of his assistants. Expenses of attendance are tax deductible.

Friday, October 31, the morning program will include an Advisors Symposium: Dr. Leo J. Starry, moderator; "The Value of This Association to Doctors," Dr. Fred Sternagel; "Office Assistants and Blue Shield," Dr. Robert L. Schaeffer; "Professional Ethics," Dr. Frederick H. Falls; "Jobs Delegated to Medical Assistants," Dr. Murray C. Eddy.

On Friday afternoon, there will be a tour of the American Medical Association Building, with a program for which the speakers, films, and exhibits will be furnished by the AMA.

At dinner, Friday night, in the Red Lacquer Room, Dr. Morris B. Fishbein will be the principal speaker.

On Saturday morning, the meeting will be divided into three sections: Business, Medical, Technical. The three meeting rooms will be fully equipped with the latest office machines, laboratory equipment, and office furniture. Medical assistants may choose those subjects in which their chief interests lie from the following:

Business: Insurance, Office Machines, Credit, Records, Telephone Techniques, Banking.

Medical: Emergencies, Medicolegal Problems, Case Histories, Good Housekeeping and Personal Grooming, Civil Defense and How We Can Help, Public Relations.

Technical: New Laboratory Procedures, New Developments in X-ray, Inter Office Professional Ethics, ECG and Ultrasonic and Diathermy, Aseptic Techniques, What is Good Practice for the Physician and Medical Assistants by Dr. Raleigh Oldfield, President Illinois Medical Society.

At the Saturday afternoon session the speakers will be: Harold Scherer, Business Manager, Monroe Clinic, Monroe, Wis., "Go and Do Likewise" and I. D. Harvey, Divisional Sales Manager, Abbott Laboratories, "Is The Welcome Mat Out."

In the evening there will be a cocktail party followed by a banquet in the Grand Ball Room, with installation of officers, presided over by Mrs. Mary Kinn, president, with Dr. Robert J. Samp, Director of Tumor Clinic, University Hospitals, Madison, Wis., as the master of ceremonies.

Sunday morning there will be a Brunch with farewell from Chicago and invitation to Philadelphia in 1959.

Hotel reservations should be made directly with the Palmer House, Chicago, Illinois. Singles $8 to $16; doubles $14.50 to $20; twins $15.50 to $22.

Reservation fee of $18 for the meeting includes transportation to and from the AMA building, dinner Friday night in the Red Lacquer Room, Saturday's cocktail party and banquet in the Grand Ball Room, and Sunday morning Brunch.

Hallie Cummins, RRL
Chairman, Public Relations Committee
American Association of Medical Assistants

CLINICAL CONFERENCE ON CANCER CHEMOTHERAPY

November 14 and 15, 1958

The third annual clinical conference for Texas Physicians will be held Friday, November 14, and Saturday, November 15, 1958, at The University of Texas M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston.

Registration: 8:30-9:00 A.M., Friday, November 14, at the auditorium of M. D. Anderson Hospital. Pre-registration would be appreciated. (Since the Rice-Texas A. & M. football game will be played at Rice Stadium in Houston, Saturday, November 15th, it is recommended that hotel reservations be made well in advance of the Clinical Conference. If interested in attending the football game, please contact the Athletic Office, Rice Institute, Houston.)

Hours: Members of the Academy of General Practice desiring credit for attendance at this conference must register in advance with The University of Texas Postgraduate School of Medicine, Jesse Jones Library Building, Houston, Texas. Eight hours will be certified for attendance of this conference. A $5.00 registrar's fee must accompany the registration for the Academy. Make checks payable to The University of Texas.

SOCIAL SECURITY FOOTNOTES

SOCIAL SECURITY SAYS: "It is common knowledge that most of us because of living costs, social standards, and economic misfortunes, do not set aside enough money or other assets during our working years to provide adequately for ourselves or our families when earned income is cut off by disability, old age, or death."

In other words: Social Security believes that "most of us" must depend upon the government in our "hour of need."

SOCIAL SECURITY SAYS: "Your social security taxes pay for these nine programs:

Social Insurance
- (a) Unemployment insurance
- (b) Old Age and Survivors Insurance

Public assistance to the needy
- (a) Old-age assistance
- (b) Aid to the needy blind
- (c) Aid to dependent children
- (d) Aid to the permanently and totally disabled

Children's services:
- (a) Maternal and child-health services
- (b) Services for crippled children
- (c) Child-welfare services

In other words: In spite of the fact that most of these represent federal grants to state aid, this Social Security program is being sold to you and me as "contributory social insurance."

TULANE STUDENT AWARDED SCHOLARSHIP

Miss Donna N. Kern of 1315 Gravier Street, New Orleans 12, Louisiana, now in her third year at Tulane University School of Medicine, New Orleans, has been awarded a $500 scholarship for research and clinical training this summer in the field of the allergic diseases.

Miss Kern will work on the histochemical demonstration of antiplatelet antibody through the use of fluoroscein-labeled Coombs' Serum, under the direction of Dr. Vincent J. Derbes, Professor of Medicine and Director of the Division of Allergy and Dermatology, Tulane University School of Medicine.

CARIBBEAN MEDICAL CRUISE

The University of Texas, Postgraduate School of Medicine announces its Second Medical Cruise to the Caribbean. Five outstanding lecturers experienced in teaching postgraduate courses have been obtained for the cruise representing fields of medicine, radiology, surgery, obstetrics and gynecology and pediatrics. The cruise (May 5 to 18, 1959) will sail from New Orleans on the M.S. STELLA POLARIS. Twenty-five hours of scheduled teaching will be offered. Inquiries should be directed to The University of Texas, Postgraduate School of Medicine, Houston 25, Texas.

ANNUAL MEETING THE TRI-STATE MEDICAL SOCIETY

The 1958 annual meeting of The Tri-State Medical Society will be held September 10 and 11, headquarters at the Confederate Memorial Hospital, Shreveport, Louisiana. The program is as follows:

WEDNESDAY - SEPTEMBER 10, 1958

8:30- 9:00	Registration — $10 for non-members, members, free Dr. Jack Guthrie—Presiding
9:00- 9:40	"Histoplasmosis"—Dr. Harris D. Riley, Jr., Professor of Pediatrics—Oklahoma University Medical School
9:40-10:20	"Allergic Conditions in Children"—Dr. Jack Anderson, Associate Pofessor of E.N.T., Louisiana State University Medical School
10:20-10:40	View Exhibits
10:40-11:20	"The Surgical Complications of Pregnancy"—Dr. Isidore Dyer, Professor of Obstetrics, Tulane University Med. School
11:20-11:30	View Exhibits
11:30- 1:00	Clinics and Case Presentations in Pediatrics, E.N.T.
1:00- 2:00	Luncheon—Free for Registrants Dr. B. H. Joslin—Presiding
2:00- 2:45	"The Not So Simple Colles Fractures"—Dr. Jack Wickstrom, Professor of Orthopedics, Tulane University Medical School
2:45- 3:00	View Exhibits
3:00- 4:00	Clinics and Case Presentation in Orthopedics and Obstetrics
6:30- 8:00	Cocktail Hour—
8PM-12PM	Social Hours at East Ridge Country Club—Dinner and Dancing

THURSDAY - SEPTEMBER 11, 1958

	Dr. R. B. Langford—Presiding
9:00- 9:40	"Cardiology"—Dr. Don Chapman, Associate Professor of Medicine, Baylor University Medical School
9:40-10:20	"Surgical Treatment of Cardio-Vascular Disease" Dr. Denton Cooley, Associate Professor of Surgery, Baylor University Medical School
10:20-10:40	View Exhibits
10:40-11:35	"Benign Disorders of Peripheral Nerves"—Dr. Bertrum E. Sprofkin, Associate Professor of Neurology, Vanderbilt University Medical School
11:35- 1:00	Clinics and Case Presentations in Medicine and Surgery
1:00- 2:00	Luncheon—Free for Registrants
2:00- 3:00	C P C—Drs. Chapmen, Cooley, and W. R. Matthews
3:00- 3:30	Business Meeting
3:30	Adjourn
3:30	Dove Hunt for all Interested

WOMEN'S ACTIVITIES

Wednesday, September 10, 1958

10:00- 1:00	Swimming Party for Ladies—Home of Dr. A. J. Nicholas 1820 Audubon Place

Doctors Note: **Please bring this to the attention of your wife.**

Please send your registration fee of $10 to Dr. J. C. Sanders, Treasurer—106 East Kings Hwy., Shreveport, Louisiana

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

ORLEANS PARISH

Membership of the Orleans Parish Woman's Auxiliary was jubilant when news was received from Mrs. F. W. Raggio, Jr., Essay Contest Chairman for the State, that Bonnie Sue Harris of New Orleans placed third in the national competition.

The top winners were: Herschel Mills of Angleton, Texas, first, and Joan Legoski of Wayne, Wyoming, second.

The Essay Contest is sponsored nationally by the Association of American Physicians and Surgeons.

IBERVILLE PARISH

The final meeting of the season of the Future Nurses Club was held in June at the home of Mrs. Frank O. Tomeny in White Castle.

An award is presented annually to the outstanding senior member of the club by the Woman's Auxiliary to the Iberville Parish Medical Society. The recipient this year was Sharon Blanchard who plans to enter training at Our Lady of the Lake School of Nursing in Baton Rouge.

Mrs. Branch J. Aymond,
Press and Publicity.

BOOK REVIEWS

Battle for the Mind; by William Sargent, Garden City, N. Y., Doubleday & Company, Inc. 1957. pp 263. Price $4.50.

This volume is concerned with the determination of the physiological mechanism involved in the fixing or destroying of belief in the human mind. In his study of the subject, the author attempts to show that consciously or unconsciously, a common physiological *modus operandi* is employed by politicians, psychiatrists, evangelists and even, in early times, by medicine men, the technique being modified by all kinds of people to suit the demands of the day and place. The author believes that an analysis of the method employed in such an indoctrination leads to future promise in the treatment of the mentally ill.

Dr. Sargent is at present in charge of the Department of Psychological Medicine at one of England's oldest and best-known medical schools. He was for many years on the staff of Maudsley Hospital in London, England's post-graduate psychiatric teaching and research center. He is president of the Section on Psychiatry of the Royal Society of Medicine and Registrar of the Royal Medico-Psychological Association. He has visited or worked in the United States on four different occasions in the past eighteen years.

MARY LOUISE MARSHALL

Halsted of Johns Hopkins, the Man and his Men; by Samuel James Crowe, M. D. Springfield, Illinois, Charles C Thomas, 1957, pp. 247, illus., Price $5.00.

This book, written at the request of the Department of Surgery, Johns Hopkins, is a history of the establishment and development of this department. Dr. Crowe was personally associated with all the men of whom he writes and gives here the details which make them live for the reader. Chosen by Dr. Halsted to work with him were Drs. Harvey Cushing, William Stevenson Baer, Walter Edward Dandy, Frederick Henry Baetjer, James Farnandis Mitchell, Samuel James Crowe and Hugh Hampton Young. Dr. Halsted is credited with establishing Residency training in Surgery and it is interesting to note that of 17 resident surgeons serving under him, seven became Professors of Surgery, one a Clinical Professor, three Associate Professors, one an Instructor and one Surgeon in Chief of a large hospital. Of the 55 assistant resident surgeons, 15 became Professors, five Clinical Professors, five Associate Professors, three Assistant Professors, five Assistant Clinical Professors and eight Instructors in Surgery. In a total of 238 surgeons trained under Halsted, 99 went into private practice and 139 into full-time University work. This book is recommended to every surgeon, every medical library and to those interested in medical education and the history of medicine.

(Sgd.) MARY LOUISE MARSHALL

PUBLICATIONS RECEIVED

Grune & Stratton, Inc., New York: Alcoholism, by Arnold Z. Preffer, M.D. Progress in Cardiovascular Diseases (Vol. 1), edited by Charles Friedberg, M.D. Developmental Potential of Preschool Children, by Else Haeussermann.

Paul B. Hoeber, Inc., New York: Clinical Obstetrics and Gynecology (Vol. 1 & 2), Toxemias of Pregnancy, edited by Louis M. Hellman, M.D. and Fibromyomas of the Uterus, edited by Robert A. Kimbrough, M.D. Milestones in Modern Surgery, edited by Alfred Hurwitz, M.D. and George A. Degenshein, M.D.; foreword by J. Englebert Dunphy, M.D.

Johnson & Johnson, New Brunswick, N.J.: Therapeutic Uses of Adhesive Tape.

W. B. Saunders Company, Philadelphia: Diseases of the Esophagus, by J. Terracol and Richard H. Sweet.

Charles C Thomas, Springfield, Ill.: A Primer in Medical Technology, by Paul M. Kraemer.